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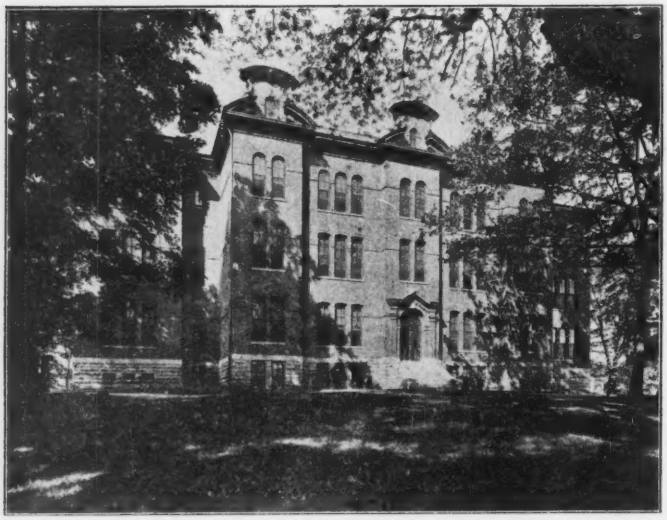

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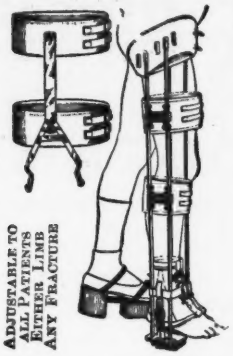

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CLINICAL NOTES ON APOPLEXY.

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In the remarks I have to make the term *apoplexy* will be used in its broadest sense, and as a general term will embrace not only the acute cerebral attack or "stroke" due to hemorrhage, but also that resulting from the plugging of a vessel. The apoplectic seizures occasionally met with in intracranial tumor or abscess, in general paralysis of the insane and in a few other conditions, will not be considered.

A "stroke" or an attack of apoplexy is vascular in origin and is due either to rupture of a blood vessel in the brain—cerebral hemorrhage—or to occlusion of a cerebral vessel—thrombosis or embolism. Hence the underlying pathology will be found in the circulatory system. With heart and blood vessels healthy a man is not stricken with apoplexy.

(1) *Cerebral hemorrhage* is due to rupture of a diseased blood-vessel in the brain, and, apart from traumatism, purpura and pernicious anæmia, probably never occurs with healthy vessels. As a result of chronic degeneration the walls of the cerebral arteries become weak and may partially give way at various points, forming minute miliary aneurisms. Able to withstand ordinary pressure, these little aneurisms are liable to rupture under excitement or sudden effort. In the great majority of cases there are associated with the arterial changes hypertrophy of the left ventricle and increased intra-arterial pressure as shown by the accentuated aortic second sound, and these changes are due to arteriosclerosis, either primary, or as an associated condition in chronic Bright's disease. In nearly all cases the rupture can be traced to three factors, viz: powerful left ventricle, high arterial pressure and weakened vessel wall. Cerebral hemorrhage is especially frequent between the ages of forty and sixty, that is, during the period when arterial degeneration is marked, while the power of the heart is still good. After sixty the heart becomes weaker, intra-arterial pressure is lower and rupture is progressively less frequent. As arterial degeneration is much more common in men than in women, so cerebral hemorrhage more frequently affects the former.

In about two-thirds of all cases the hemorrhage takes place in the internal capsule or *basal ganglia;* next most frequently in the cortex, and less often in the pons and cerebellum. When hemorrhage occurs in the brain tissue the blood tears and disorganizes the brain substance and soon forms a clot around which there is considerable œdema. This clot exerts sufficient pressure upon the adjacent brain tissue to impair or destroy its function, but the œdematous and compressed tissue will resume its function when the pressure is removed. Hence, the immediate and temporary effects of a cerebral hemorrhage are usually much more severe and extensive than the permanent ones, but as the absorption of the clot is slow the improvement in the symptoms is necessarily slow and gradual. As the œdema about a hemorrhagic lesion

often subsides early there may be noticed a rapid improvement, which is, however, only partial and due to the relief of the pressure caused by the œdema. Of course, the fibers that have been destroyed and have degenerated never resume their function, hence in all cases where the hemorrhage has been at all extensive there will be some permanent defect.

(2) *In Cerebral Thrombosis* there is occlusion of an artery due to the gradual constriction formed by obliterating endarteritis or to the formation of a clot upon the roughened wall of the vessel. When the occlusion is complete nutrition is at once cut off from all the brain tissue supplied by the plugged vessel, and function is at once suspended in this area, but when the collateral circulation is established the function returns in all parts not permanently deprived of blood. Hence as in hemorrhage the immediate effects of the stroke may be extensive and severe, but in thrombosis the improvement may be so marked that but slight permanent defect remains. As the collateral circulation is usually established within a week the improvement which occurs in thrombosis or embolism is much more rapid than that seen in hemorrhage. Thrombosis is relatively much more common in the cortical arteries than in those of the base, hence the frequency of localized and incomplete palsies in this condition. In the cortex the collateral circulation is extensive, but in the arteries of the internal capsule and base it is very imperfect as the arteries are terminal. Hence there is much less recovery in lesions of the basal arteries than in those of the cortex. Sometimes the middle cerebral artery itself is plugged, in which case there will be wide-spread softening. When the clot forms in the vessel it extends backward to the next branch, and if a portion of it should be separated at this point it will be carried into this branch, plugging it and causing another attack with more extensive paralysis within the next day or two. Oedema rapidly appears in the brain territory supplied by the occluded vessel, but it subsides as the collateral circulation is established. The ultimate effect of thrombosis is softening from fatty degeneration in the area permanently deprived of blood with descending degeneration in all the fibers involved.

Thrombosis occurs in obliterating endarteritis from any cause. In the young, syphilis is responsible for the majority of cases of apoplexy; in the old, senile endarteritis is the underlying factor. In such cases slowing of the blood current from weakening of the heart may result in thrombosis, hence the stroke often appears during sleep or after exhaustion or fatigue.

(3) *In Cerebral Embolism* the artery, which is usually healthy is occluded by an embolus coming from the heart or large arteries, and is met with most frequently in cases of endocarditis in young persons. The pathological changes are exactly similar to those met with in thrombosis.

Though the actual pathological conditions that cause the apoplectic attack in hemorrhage, thrombosis and embolism are clear, well defined and distinct from one another, the immediate effects on the functions of the area of brain involved are so nearly alike that clinically it is often impossible to determine the exact nature of the lesion. Inasmuch as the treatment of a hemorrhagic attack is practically the opposite of that of the stroke of thrombosis, the differentiation of these two conditions is of the greatest importance. Unfortunately, however, it is impossible in many cases to determine whether the attack is due to hemorrhage or to occlusion of a vessel. In hemorrhage and embolism the attack is sudden because the accident to the vessel is of sudden occurrence. In thrombosis, though the narrowing of the vessel may have been gradual, the actual closure is usually quite sudden and the attack may be as rapid in its onset as in hemorrhage or embolism.

In differentiating the three conditions there

are some features sufficiently characteristic as to possess considerable diagnostic significance though not one of them alone can be considered as conclusive. The chief points of diagnostic value are: age of patient, the existence of certain diseased conditions, exciting cause of an attack and time of occurrence, premonitory symptoms, suddenness of onset and nature of attack, unconsciousness, extent of paralysis and location of lesion, aphasia, reactionary fever, and rapidity and extent of recovery.

Age—Hemorrhage is most frequent between the ages of forty and sixty, thrombosis under forty and again in old age, and embolism in the young. The great majority of attacks of apoplexy occurring under forty and over sixty are thrombotic in origin.

Existence of Disease—The presence of arteriosclerosis, with cardiac hypertrophy, accentuated aortic second sound, high intra-arterial pressure and possible urinary evidences of chronic interstitial nephritis affords the conditions necessary for the occurrence of hemorrhage. Hence, if a man past middle life presents these signs and is suddenly stricken with apoplexy, we have strong presumptive evidence that there has been a rupture, especially if the attack occurred during excitement or physical exertion.

The existence of evidences of syphilis or of senile arterial degeneration, especially if associated with low blood pressure and weak heart or anæmia, would strongly favor thrombosis. Syphilitic endarteritis with thrombosis is undoubtedly the cause of the great majority of strokes under middle life.

A stroke in a young man or woman who presents signs of disease in the valvular region of the heart or in the aorte is probably embolic and this becomes almost a certainty if syphilis and Bright's disease can be excluded.

The exciting cause of an attack may be of distinct value when it can be determined. A stroke that occurs during the period of high arterial pressure and forcible heart action that are associated with mental excitement or unusual physical effort is very likely due to hemorrhage. It must not be forgotten, however, that rupture may take place when no such circulatory excitement is apparent. As the clotting in the vessel is favored by a feeble current, thrombosis is most likely to occur when the cardiac force is weak and the intra-arterial pressure low, hence the apoplexy of thrombosis frequently appears during sleep or during the exhaustion that follows fatigue, excitement or illness. The time of occurrence of embolism is probably of little, if any, value, in diagnosis.

Premonitory Symptoms—In *hemorrhage,* as a rule, there have been no premonitory symptoms of any value. Headache, vertigo, mental confusion and nausea are absent. There have been no slight attacks of vertigo or mental confusion followed by local paralysis and in nearly all cases the attack comes on suddenly without any warning. Likewise in *embolism,* premonitory symptoms are absent, though rarely a patient may have suffered from embolism in some other vessel, as for example, in the central artery of the retina producing sudden blindness, in the spleen or kidneys. It is especially in *thrombosis* that premonitory symptoms are common, though not always present. These prodromata may have extended back over weeks or months. The most significant symptoms are headaches, especially if induced by fatigue, mental or physical (in syphilitic cases the headache is usually worse towards night), attacks of vertigo coming on after effort or after a full meal, insomnia, mental confusion, loss of memory, numbness and paralysis. Numbness, if persistent or frequently recurring, and always referred to the same arm, leg or side, is a common symptom in these cases, and if associated with distinct signs of endarteritis is very significant, indicating as it does an abnormal state of the circulation in a localized area of the brain. The patient may have suffered from slight attacks of

paralysis, localized to one arm, leg or one side of the face, or he may give a history of a temporary aphasia, followed later by paralysis in a limb. The paralysis in these cases is often slight; it may be discovered on rising in the morning, or it may appear after a few days of headache, vertigo or mental confusion. An attack of apoplexy in a man who gives such a history is almost surely due to thrombosis.

Mode of Onset—In hemorrhage and embolism the attack is so sudden in its onset that the patient often falls stricken to the ground, the paralysis being complete at once. The attack in thrombosis may be quite as sudden, but usually the patient is conscious of a numb or dead feeling in the affected limb and he may be confused or dazed for some minutes or even longer before the loss of power is observed. In some instances the loss of power at first is localized to part of one limb, then gradually extends to the whole limb and perhaps to the whole side, but some hours may elapse before the full extent of the paralysis is shown.

Loss of Consciousness is a most valuable symptom in differential diagnosis. In *hemorrhage* it is nearly always present, appears suddenly at the time of the attack, is usually complete and as a rule lasts for at least twenty-four hours, often much longer. In very slight hemorrhages the symptom may be absent, but when the lesion is sufficient to produce a complete though slight, hemiplegia, it is pretty certain to be present, and a marked hemiplegia from hemorrhage is, according to Russell, always attended by loss of consciousness at the time.

In the majority of cases of thrombosis there is no loss of consciousness, or if present it is incomplete and of short duration. The patient is, however, often dazed and confused for a few minutes or a few hours, but consciousness is not lost. His cerebration is slow and imperfect, but he can be partially roused and responds to questions or commands. Even in a complete hemiplegia from thrombosis, loss of consciousness is unusual and a complete hemiplegia, gradual or sudden in its onset, without coma means thrombosis.

In embolism consciousness is not usually lost. The coma that is seen in some cases is, as a rule, incomplete and of much shorter duration than that of hemorrhage and when recovered from, it passes away rapidly.

The *extent of the paralysis* may be of considerable value in differentiating hemorrhage from thrombosis. In the majority of cases of apoplexy from hemorrhage there is complete hemiplegia. It is decidedly unusual to meet with a monoplegia in hemorrhage. On the other hand thrombosis often affects the smaller cortical branches, producing a paralysis of limited extent or the loss of power appears first in one limb and later in another. Excluding hemorrhage the result of traumatism, a cortical lesion is nearly always thrombotic or embolic, so that if it can be established that the lesion is cortical it is far more likely to be due to thrombosis than to hemorrhage. In favor of a cortical lesion are the following points: the paralysis may be limited to the face, to the arm or leg, or if there is hemiplegia it is incomplete, one limb being much more affected than the other. Aphasia is also a symptom of a cortical lesion, as damage to the internal capsule will probably not produce complete aphasia. Hence, a true aphasia is much more frequent in thrombosis than in hemorrhage.

Reactionary Fever and Other Signs—On the first day of the attack in hemorrhage the temperature is usually subnormal, but this is followed by a rise to 101 to 103, occasionally higher. In thrombosis and embolism, as a rule, there is no initial fall and little or no subsequent rise in temperature. The pulse in hemorrhage is full, of high tension, slow and often irregular; in thrombosis it is normal or weak and of low tension; in embolism it is the pulse of the cardiac condition. The pupils in hemorrhage are often irregular and

do not react to light; in thrombosis and embolism they are usually regular and react to light.

Rapidity of Recovery—In hemorrhage, recovery from the paralysis is slow and some permanent defect always remains. In thrombosis, and likewise in embolism, there is often marked improvement within the week, sometimes in the first two or three days, that is, as soon as the collateral circulation is established. Occasionally in thrombosis there is a marked return of power on the second day followed on the third day by an increase in the extent of the paralysis. This may be due to extension of the thrombus into other branches, but in many instances is the result of temporary vascular changes with œdema, the symptoms improving again in a few days.

Prognosis—I shall not attempt to discuss the various indications which may assist us in predicting the outcome of a case. An attack of apoplexy due to cerebral hemorrhage is much more likely to prove fatal than that due to thrombosis or embolism, in fact some of the English neurologists claim that the great majority of hemorrhagic apoplexies are fatal. We all recognize the grave import of deep and prolonged coma, of stertorous breathing, etc., and in cases presenting such symptoms we are rarely mistaken in predicting an unfavorable termination. But in cases where consciousness is not entirely lost, though hemiplegia may be complete, prognosis is so difficult that the only safe ground is to withhold an opinion and await developments. In thrombosis or embolism the collateral circulation may be quickly established and a remarkable improvement in all the symptoms may be seen even as early as the second day. On the other hand, the later development of œdema or extension of the thrombus in an apparently mild case may cause an aggravation of the symptoms with coma and a rapidly fatal termination. In thrombosis the third day often seems to be critical and probably coincides with the development of extensive œdema in and about the area of brain supplied by the occluded vessel. Again in thrombosis one must not forget the danger of recurrences. Even in syphilitic cases this is, I believe, an ever-present danger, for when cerebral syphilis has advanced to the apoplectic or paralytic stage, I have but little confidence in complete cures by even large doses of the iodides. It cannot be expected that these drugs will restore degenerated tissues to their normal condition.

DIAGNOSIS OF PAINFUL FEET.

BY E. LAURENCE SCOTT, M.D., ORTHOPEDIC SURGEON TO HILLMAN HOSPITAL, BIRMINGHAM, ALA.

In the consideration of some of the more common conditions giving rise to pain in the foot, it will not be my intention to dwell on the pathology of any one particular condition, but rather to try and bring to your notice the symptoms upon which we may rely for our diagnosis.

I think that I am correct in believing that to the majority of the general practitioners of medicine, the words "painful feet" mean one of two things—that the patient has either flat-foot, or that he has rheumatism.

It is not usual for one in the general practice of medicine to make a careful and painstaking examination of the foot and, therefore, the foot in the majority of cases is not recognized as a part of our anatomy whose function is peculiar to itself.

Painful feet probably constitute the larger portion of the cases coming to the notice of the orthopedic surgeon, and I will therefore follow out the line of diagnosis as used by these surgeons.

The human foot in general outline may be roughly likened to the ordinary span bridge. At each end of the foot is a support or pillar, and the intervening part, or body proper, reinforced and held in place by ligaments and fascia, and the muscles approaching the foot from the leg.

At the posterior end of the foot we find the os calcis as the supporting pillar, and at the anterior end the heads of the five metatarsal bones with their respective phalanges serve the same purpose. The outer edge of the foot is reinforced or braced in its proper position by the fifth metatarsal bone, which acts to prevent the foot from rolling outwards when the body weight is borne. The support of this bone is, however, of no great consequence and does not deserve particular attention.

The body of the foot is composed of the tarsus, consisting of the os calcis, already mentioned, the scaphoid, the astragalus, the cuboid, and the three cuneiform bones.

The metatarsus, which is the anterior segment of the foot will be described in a later part of this paper.

The muscles approaching the foot from the leg, play the same part in the support of the arch of the foot as do the cables in a bridge.

When thought is given to the fact that the foot bears more weight and is subject to more strain than almost any one part of our body, it will be easy to appreciate and understand the rapidity of evidence of strain or perverted function here.

It is on account of the very completeness of the structure of the foot that we find such marked exhibition of symptoms when any one bone is slightly displaced or any ligament strained. Each separate part of this complete structure depends, as it were, on the other, in the performance of the function that is intended, particularly so during locomotion, and the taxation on the foot as a whole from the superimposing of the body weight.

The consideration of the condition known as FLAT-FOOT would probably be suggested first, in a paper dealing with the diagnosis of painful feet.

What do we mean by the term flat-foot? Do we mean that a foot is literally flat, as the term would indicate, or do we mean a foot which is undergoing the pathological changes that will eventually result in such an anatomic formation? Every one has seen a foot which is flat, but that foot may not be a painful one, and so never come to treatment, save possibly for an improvement cosmetically, or to seek relief from an awkardness in the gait. This is not the type with which the

general practitioner usually has to deal, and an omittance of anything more than the mere mention of this type will be necessary on account of brevity.

Recent writers on painful conditions of the feet, have emphasized that our accepted standard of highness, or classic appearance of the arch of the foot is subject to wide variations and that we may find all grades of highness or lowness of the arch, both longitudinal and anterior arch, and that in the particular individual, it may be the normal. In view of these statements, the question of standard height or lowness, seems to resolve itself into a personal equation, and we must consider the height or lowness of the arch as peculiar to the person under examination only, and to bear weight in the diagnosis accordingly.

It is not so much the degree of lowness in the longitudinal arch or the displacement of the tarsal bones that characterizes the foot as a pathological or *symptomatic* flat-foot, as it is the amount of resultant strain to the ligaments and muscles entering into the foot. Whitman, of New York, has very aptly put it that "Pain is not caused because the foot is flat; it is a symptom of strain and injury, and of progressive deformity."

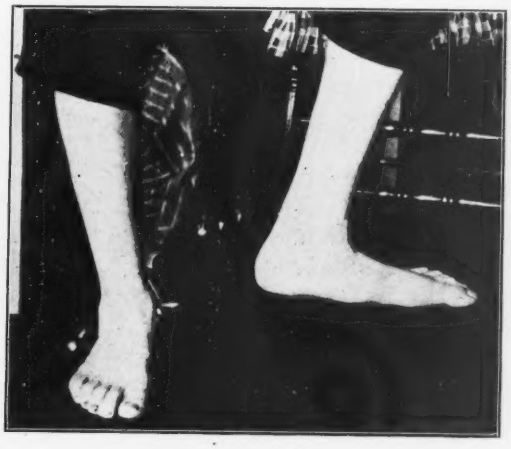
In the class of cases known as weak feet, a person may present a high and classic arch and still suffer acutely from the slightest strains or displacements, which displacements may be of such small degree as not to be detected in the ordinary casual examination.

The external appearances of such feet sometime confuse one to explain the severity of the symptoms, and the fact that the slightest disturbance of the proper relationship of the bones of the tarsus will call forth decided symptoms, is quite often overlooked.

The STATIC FLAT-FOOT is a type mostly coming to our notice, and is a result of occupational usages, or better, abuses, and is mostly seen in persons who stand or impose the body weight on the feet for long periods at a time.

This variety of painful foot is found most frequently in persons whose occupations are such as motormen, shop girls, and in those who are not able to rest the foot and relieve them of the strain of weight bearing at frequent intervals.

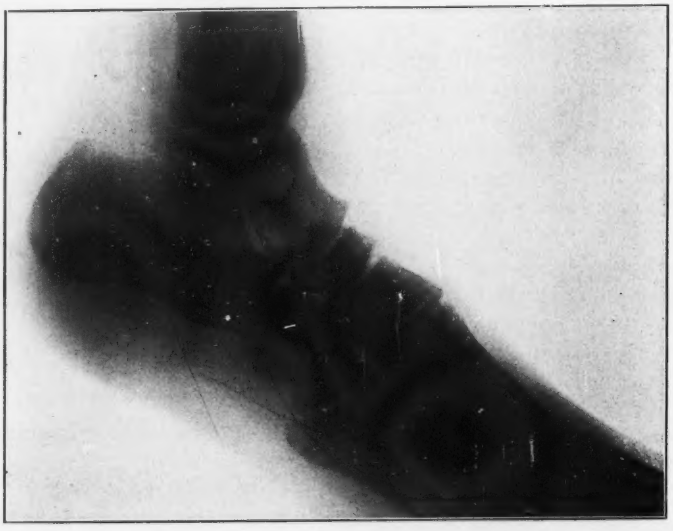
No. 1. Typical flat foot - "non-painful." Showing the pronation and turning outward of the front part of the foot causing a shambling gait.



No. 2. Same case as No. 1. No weight bearing. Showing the downward displacement of the Scaphoid and destruction of the norma inner contour of the foot and obliteration of the arch.

The history of these cases is generally clear as to the commencement of the trouble, and as to the mode of onset. They usually tell that the foot at first gave a sensation of uneasiness and tired quickly. As the deformity progressed, a decided sense of discomfort was experienced, and then they became conscious that there was something wrong in their regular gait, and that they were accustoming their steps to suit their feet and placing the foot carefully, avoiding any unevenness in the walk. A dull, aching pain is felt in the foot, be caused. The foot perspires freely, although no rise in surface temperature be found.

When the foot is at rest the natural elasticity of the ligaments and fascia, cause a partial replacement of the tarsal bones, which gives comfort, but when the foot is again put into activity, the degree of discomfort seems increased. This is of course to be accounted for by the renewed strain to the ligaments. This of itself is sometimes enough to cause the patient to dread to put weight on the foot, and in some cases it is several minutes before



No. 3. Radiograph of foot shown in No. 1. Illustrating the effect on the tarsel bones, of changed relation of the Os Calcis, to the Astragalus.

at first not confined to any one particular point, but later the points of strain can be recognized. The pain may in some cases extend up into the calf, the knee or even to the hip. The foot may be swollen and is sensitive to pressure at such points as the insertion of the plantar fascia, and at the articulation of the scaphoid with the astragalus.

The normal amount of flexion, extension and adduction and abduction is limited, and when such movements are attempted pain may a step will be attempted, and then in a most guarded manner.

Besides the careful consideration of the symptoms exhibited it is necessary to make a careful general examination as to the weight bearing lines, the contour of the inner border of the foot and the manner of walking and placing of the foot on the floor. To make such an examination the patient should stand with feet bare, heels approximated, and toes turned slightly outward. Weight should be

borne *equally* on each foot. It is important that the weight be equally borne, for in some cases an inequality in this particular will give a very different picture than that which really exists during equal weight bearing.

A static flat-foot in most cases will show some degree of pronation, varying of course with the duration and severity of the condition. The tendo-achilles in the normal foot comes to its insertion into the os calcis in a straight line, but when pronation is present, this tendon makes a sweeping curve, with the convexity of the curve to the median line of the body.

A test of the flexion, extension, adduction and abduction powers, as above mentioned, should be made, for in some cases we may find the pronation due almost entirely to a spasm of the peronei group of muscles.

The internal border of the foot should be inspected, to detect any downward and inward displacement of the scaphoid or rotation of the astragalus.

Everyone is familiar with the impression as made by the average foot, bearing weight, and is quick to recognize any deviation from the normal, so that I shall only mention the method I think a good one, and not go into the merits of this manner of aiding the diagnosis, because, as said before in referring to the height of the arch, the foot may be normal for the patient under examination when it would appear not to be so. It is necessary that an impression be taken of both feet, even though only one foot may be affected and in case both feet are affected the physician must use his judgment in reading the impression. The method of impression-taking as used by Frieberg is a good one, and is as follows: The sole of the foot is moistened with a solution of chloride of iron and imprint stained on paper. By immersing this paper in a tannic acid solution, the stain becomes a durable black. One value of the impression is that it can be used for future references in the treatment and to show improvement from such treatment as is given.

I have used the term static flat-foot, and the term weak-foot, interchangeably, for two reasons, first that space is not allowed for separate consideration, and secondly, that one type does not exist entirely without the other, as can be readily seen. The class of cases known as *rigid* flat-foot do not constitute a large enough proportion of the cases coming to the notice of the general practitioner to deserve special attention.

I have taken up the time allowed me on affections of the longitudinal or greater arch in the diagnosis of a typical case and allowance must therefore be made for the class of cases presenting the intermediate grades of severity, as seen in the simple weak foot or in the simple pronated foot.

A condition of second importance to relaxations and affections of the longitudinal or greater arch of the foot is found not infrequently in the anterior arch. This condition is probably known to most as MORTON'S TOE, OR MORTON'S NEURALGIA, on account of attention being first called to the condition by Dr. T. G. Morton, of Philadelphia, in 1876. Some more recent writers give to the affection the term "anterior metatarsalgia.

In the foregoing part of this paper, the anterior arch was mentioned as the anterior segment of the foot, and as the anterior support of the bridge. Nothing was said of its peculiar anatomy, so therefore a brief review of its bony architecture will be necessary for an understanding of the condition I shall mention. This anterior metatarsal arch is formed by the bodies and heads of the five metatarsal bones, held in place by the interosseous muscles and ligaments with the exception of the first metatarsal bone, which is analogous to the thumb, through not being connected to its neighboring bone by ligaments.

Counting from the inner side of the foot outwards, it will be found that the second and

third metatarsal bones are longer and occupy a slightly higher plane than the remaining ones and it is due to this peculiarity almost entirely, that the use of the term arch is justified as descriptive of this segment of the foot. This arch exists as an arch only when the foot is at rest and when no weight is borne. A great deal of elasticity and resiliency is possessed here, and the arch formation is resumed in the average person as soon as the pressure of the shoe or the weight of the body is removed from the foot.

The movements, upward and downward, of the metatarsal bones as occurring during walking, does not occur simultaneously for all, but is more of a succession of movements, starting first in the fifth metatarsal bone and going across to the first, as the step is made. This is true in persons who place the foot in the proper position for weight bearing, or in other words, when the degree of *Abduction* of the foot is the minimum.

This upward and downward movement is very free, and may so account for the symptom found in affections of this arch. The pathology of a typical affection of this kind has not been fully demonstrated, nor has the true etiology been satisfactorily explained. Several explanations have been offered from time to time, but as none seem conclusive, a review of them would avail nothing of interest.

The symptoms, or rather symptom, usually corresponds in the average series of cases seen, and is practically diagnostic in itself. The characteristic cramping pain across the ball of the foot, as the patient will put it, usually gives us the diagnosis, though in some cases the pain may not be so definitely located. The acute pain is felt mostly between the third and fourth toes, or between the fourth and fifth toes. This pain may radiate into the distal ends of the phalanges, or go even into the calf of the leg. It is not a constant pain, but is intermittent, and rarely comes on except when the shoe is worn or when weight is borne on the foot. The frequency and severity of the pain may be increased by wearing a tight shoe; the converse of this is equally true. The neuralgic like pain may in some cases be found, but whether it be a cramping or neuralgic type, it is usually relieved by removing the shoe and manipulating the front part of the foot. The intermittent character of the pain, and its acute onset, should warn us in our diagnosis of a

No. 4. Radiograph of foot giving symptoms of Morton's toe, illustrating mal-position of the second and third metatarsal bones, which pinch against each other (at point marked "X") when the anterior arch is compressed laterally.

Morton's toe, for in the discomfort due to an ordinary relaxation of the anterior arch, the pain is almost constant, and appears in the slightest weight bearing, and is not so severe. Again, in general relaxation, with resultant strain, the point of pain is not so definitely localized, and the feeling is more one of tiring and aching, than of acute cramping.

In Morton's toes, a general examination of the foot will probably show nothing abnormal, whereas in the static relaxation, callouses will be found under the heads of the metatarsal bones. The use of the Rœntgen ray for a diagnosis of affections of this portion of the foot, is practically valueless, save to clear up any possible doubt that may exist as to the presence of exostosis, or of mal-positions of the

No. 5. Radiograph of foot with symptoms identical to No. 4.

metatarsal bones due to the compression of faulty foot-wear. Malpositions, giving rise to pain from the rubbing of one bone against its neighbor, is in my opinion, not of uncommon occurrence.

The subject of GOUT would probably not be warranted in a paper dealing with the diagnosis of affections of the foot alone, save for the fact that the primary manifestations of this condition are to be found in the foot, in the larger percentage of cases. I shall not take up time in the consideration of the symptomatology of the chronic form of gout, but shall confine myself to the acute form.

The pathology of gout is well described in most text-books on the practice of medicine, and is easily understood by all. Gout usually manifests itself first in the metatarso-phalangeal joint of the great toe, and the symptoms as a rule come on in the early morning hours. The pain, which is peculiar to the affection, is described by those who have experienced it, as if the foot or rather the metatarso-phalangeal joint were compressed in a vise. The signs of inflammation quickly supervene, the heat, redness and swelling in some cases becoming very pronounced. A rise in the body temperature nearly always accompanies the acute attack.

The attacks depend for their frequency and severity on the habits of the patients, being increased in frequency through indescretions of the diet, etc. The duration of the attack is usually not long, and the acute symptoms subside with a copious perspiration. Some difficulty may exist in making a diagnosis between acute gout and acute articular rheumatism; the main point in the diagnosis is the recognition of acute articular rheumatism being polyarticular, while gout is primarily confined to the articulation above mentioned. The chronic form as diagnosed from the chronic form of articular rheumatism is more difficult, and requires a careful examination and history-taking, but again we know that a chronic rheumatism has a predilection for the smaller joints, and chronic gout for the larger.

In the differentiation of gout from the variety of arthritis known as the *hypertrophic*, and the form known as *atrophic*, it is to be remembered that gout is rather sudden in appearance, while the other two are slow and insidious.

Gonorrhœal Arthritis is frequently seen in the foot, but the diagnosis is as a rule not dif-

ficult, when the history of the case is concise and taken carefully.

Baer, of Johns Hopkins, has written on this subject and has demonstrated the gonococci in the scrapings from the periosteum of the cases that came to operation.

This type of arthritis may not come on until after the subsidence of the acute attack of gonorrhœa, and with a certain class of patients, a denial of any such infection may be given, when the case will of course be more difficult of diagnosis, and the diagnosis will have to be one, more of suspicion than of proof, unless the case comes to operation, when, as above said, it may be possible to demonstrate the actual presence of the gonococcus. The symptoms seen in these cases are about as follows: Sudden pain in the arch of the foot, swelling, redness, and rise in temperature. Acutely painful points may be found on pressure, and are to be looked for in such places as the under surface of the os calcis, the region of the astragalo-scaphoid articulation, or in some, under the insertion of the tendo-achilles into the os calcis.

The use of the Rœntgen Ray is valuable in this affection, on account of the formation of exostoses, which can be plainly seen in a good radiograph. The under surface of the os calcis is the most frequent location of the exostoses, but they may be found at almost any articulation in the tarsus, or at the insertion of the tendons coming into the foot..

ACUTE ARTICULAR RHEUMATISM, confined to the foot alone, is not of common occurrence. This is probably to be explained by the fact that the joint architecture of the foot is not the same as is found in the other joints of the body, with the exception of the wrist. It is more of a series of smaller joints with little or no true capsule formation.

It is not uncommon, however, to find this affection in the foot secondarily, and then we may see the affection in the worst phase, resulting in contractures and disabilities of a permanent nature.

It is almost unnecessary to go into the symptomatology of acute articular rheumatism, as nearly every one is familiar with the condition as it is seen every day, so it has been my aim and attempt in this brief paper to try and divert attention from acute articular rheumatism, and at the same time to show that the foot is subject to a goodly number of conditions, resulting in pain and disability, and that we do not owe so much to the disease, acute articular rheumatism, in the diagnosis of painful feet, as has been supposed.

Summary: First—Acute painful flat-foot is found in persons who subject their feet to abnormal strains. Weak feet, speaking generally, are found in persons who do not accommodate the foot usages and in those whose general bodily condition is below the normal. Weak feet, if untreated, may result in true flat-foot, or chronic strains and displacements in the structure of the feet. Diagnosis is usually not difficult and is made by exclusion unless the evidence of perverted function be plain.

Second—Morton's Toe, or anterior metatarsalga is most frequently seen in women. The cramping pain, in the region of the metatarsophalangal joint of the third or fourth toe gives the diagnosis. The pain from the strain resultant of relaxations and that due to malpositions and exostoses, is not experienced except when foot is in activity, while in the first mentioned condition, the pain may appear even during rest.

Third—The diagnosis of gout must always be one, more of suspicion than of proof, in the average case the condition must be distinguished from the other types of arthritis. The diet, and habits of patients bring on attacks and so this is the leading point in diagnosis.

Fourth—Gonorrhœal arthritis affecting the foot is in some cases difficult of diagnosis on account of denial of infection. The formation of the exostoses on the os calcis and the other tarsal bones makes it clear. As would be inferred, males are most frequently subject to this condition.

A NOTICE OF SOME OF THE RECENT WORK ON STOMACH DIGESTION.

BY W. H. WITT, A.M., M.D.

The study of physiology and the conduct of physiological experiment, while interesting in themselves and at times even fascinating, do not have the earnest sympathy of the practicing physician or surgeon for the reason that it is often a far call from normal physiology to the treatment of pathological conditions and the relation of one to the other is not always clear, and the full understanding of the one too often seems to shed little light on the other. Probably the great bulk of our therapeutic knowledge has come to us in an accidental way instead of by logical deductions from physiology on the one hand, or physiological action of drugs on the other. But we are coming to know more of the real secrets of physiology and some very happy applications have been made of our recent knowledge. The physiology of digestion has always had its devoted students, and in recent years no less skillful ones than in the years before; for by the labors of Pawlow in Russia, Bayliss and Starling in England, and Cannon and others in America, we have had real acquisitions to our knowledge of this important function. And inasmuch as all of us are disposed to neglect reading matter of this kind, I thought it might not be out of place to bring to our minds the gist of this work and see wherein it confirms established practice, and wherein it may offer suggestions of untried methods. I may say to start with that a good part of recent demonstrations serves to confirm clinical observations and preconceived notions and put them on an established basis of proof, and to that extent some of what I shall say may not appear at all new.

Physiologists are indebted to Pawlow for a very ingenious method of studying gastric secretion. He conceived the idea of diverting a portion of the stomach in such a way that the diverticulum, while not communicating with the general cavity of the stomach, yet did not have its nerve or blood supply interfered with. By this device he could study gastric secretion under the influence of nearly all the stimuli that normally obtain in promoting that function. Pawlow proved that simple mechanical stimulation did not cause a secretion of gastric juice, but of an alkaline mucus —in effect a protective secretion. He also showed that alkaline solutions instead of promoting the flow of gastric juice—as had for so long been believed and taught—in reality retarded it. I think this finding rather confirms clinical conclusions in reference to the use of the alkalies as a promoter of stomach secretions. In old times, before stomach analyses were made, cases of high or low Hcl secretions were not properly differentiated, and alkalies being helpful in many cases of dyspepsia and promoting stomach comfort and appearing to promote stomach digestion, it was concluded they were in effect stimulants to the flow of gastric juice. Clinically, while alkalies give relief from the symptoms of hyperchlohydria, I do not believe that the inhibiting effect Pawlow has found is a very great factor in effecting a cure. Their value is chiefly to give temporary comfort.

Pawlow's most effective work was to put on a rather solid basis of proof the various agencies that stimulate the flow of gastric juice. Under ordinary circumstances the secretion is largely a reflex act. Dogs were tested in various ways, and it was found that the sight of food or its smell were sufficient to cause a ready flow, but that the secretion was best stimulated through the sense of taste and as a reflex from mastication, especially of foods that have some pleasant savor. This reflex secretion which Pawlow called the appetite

juice could be kept up rather indefinitely if the hunger of the dog was not appeased. He effected this by dividing the esophagus in the neck and drawing the distal end of the upper portion to the surface so that when the dog swallowed the food it would not reach his stomach. It was also noted that the secretion of the gastric juice would often continue much longer than the actual presence of the stimulus. Hornburg made similar experiments on a boy who had an esophageal stricture with a gastric fistula. He and others observed, too, that the mastication of rubber or other indifferent substance failed to excite the secretion. In fact, throughout these experiments it was noted that the savory quality of foods had a large influence on the amount of secretion. Hunger was always found to play an important part.

From these findings we get several suggestions of value in feeding—but they are for the most part things that we have known—simply put on a more rational basis. In the first place in so-called weak stomachs frequent feeding of small amounts is justified by Pawlow's observation that in this way a large gross amount of secretion was secured in the 24 hours. Delicate stomachs and poor digestion for all time been treated this way, but on the ground, I think, that a fuller meal overtaxed the stomach physically, or transcended its digestive power. Now we are taught that while small feedings are better, because larger ones may be rejected or give pain—they are also better because they promote a more continued secretion.

Pawlow's experiments also confirm the accepted opinion that foods that have a good relish are most easily digested—though we know, too, that there are some very marked exceptions to this rule and its too close application in practice would lead us into some bad mistakes, for the more savory foods, and those we like best, are not necessarily the most digestible. In fact, a food such as meat, may have a good taste and tend to promote secretion of hydrochloric acid, and yet its digestive requirements be much higher than milk, for instance—that to many people is insipid. But even the more bland foods such as milk and eggs can often be so prepared and served to a patient as to be appetizing, and thus offer a stimulus to stomach secretion. Apart from flavoring milk or cooking it with other food stuffs, it seems possible to promote its digestibility by such simple measures as sipping it or adding carbonated water.

Again, the value of thorough mastication receives fresh support from the proved fact that mastication is an active agent in causing the secretion of the appetite juice. The relish of foods and their thorough mastication are in this matter closely associated. Pawlow found, too, that the much declaimed against meat extracts have their use, though their calorie value is very small, in the fact that they are often relished when the more insipid things are not, and they may prepare the stomach for digestion of less appetizing dishes by their stimulating effect on the secretion. The service they perform is a genuine one, but should not let us forget that they are not of much value as foods. The suggestions furnished, namely, frequent small feedings, appetizing foods, and thorough mastication apply to a rather large number of cases that might rather be called feeding cases than stomach cases—for the reason that the deficient secretion and deficient appetite are a result of constitutional conditions such as cancer, nephritis, anemia, tuberculosis, neurasthenia, etc. I think it is rather rare that we find a deficient Hcl secretion except as a part of a more widely extended pathology—or at least a functional disturbance that applies to the stomach only as a part of the system. But the hope for improvement of many of these patients, especially the tuberculous, anemic, and neurasthenic lies in our success in giving them nourishment. As to the use of bitters, Pawlow approves them, but holds

Beside alkalies, to which I have referred, Pawlow observed that fats inhibited very distinctly the secretion of Hcl, and when given with proteid were able to retard the digestion that they act only by improving the appetite.

of the latter. Clinically, the disturbing effect that certain fats, especially meat fats, have on stomach digestion, is one of our commonest observations. Milk fat, butter, cheese, etc., retard much less. What light is thrown on the treatment of an over secretion of Hcl-hyperchlorhydria? Understanding by this term, glass of milk or most any kind of food. They too, that there is not necessarily a high percentage of the acid, but that the essential factor is rather a hypersensitive mucous membrane and that the Hcl may be normal or even a little low. There are the patients that have pain when their stomach is empty—that get relief by water acting as a diluent, or by a have good digestion and a clean tongue. We give them alkalies that neutralize, and according to Pawlow inhibit to some extent. Or we say take a glass of milk or water when pain comes on or eat a little of most anything. All of these give relief temporarily. From what I have said, however, frequent eating and having a tendency to keep up the secretion of Hcl is contraindicated. Even water is said by Pawlow to act as a real stimulant to Hcl secretion. The definite indications growing out of the foregoing statements would, in hyperchlorhydria, be about as follows:

Two or three full meals instead of frequent meals; these meals to be of the less savory foods and to contain a good proportion of fats; as much insipid stuff as can be used. As to meats: Proteid takes up a large per cent. of Hcl, and is to that extent indicated, but as a well-known stimulant to secretion of Hcl. it is contraindicated. Theoretically, large amounts of good steak and roast would seem to be bad, though in practice they are recommended by many specialists. Condiments and relishes are not to be used. Sweets inhibit Hcl. (Pawlow). This kind of diet would seem to meet the indications because it tends to rather lessen the amount of secretion, though clinicians vary very widely in their management of this functional disturbance. For the pain, alkalies to neutralize and water to dilute. Incidentally, many of these patients are to some extent neurotics and treatment of that feature is called for.

In addition to the gastric juice secreted in response to nervous stimuli there is a second secretion noted to occur when all connection of stomach with the central nervous system both by the pneumogastric and sympathetic is severed. It is proven, too, that this secretion is not due to mechanical irritation of food nor does it occur with a number of foods such as white of eggs, starch or bread, when they are introduced into the stomach, but does occur if they have been previously partly digested and then put into the stomach. But meat, meat extracts and milk and many other foods provoke this secretion when put into the stomach through a tube. It seems proven that this secretion does not occur as a purely local reflex by way of nerve plexuses in the gastric wall, and it is now accepted that meats, meat extracts and milk and other foods when first introduced, and others when partly digested contain certain chemical substances that act on the mucous membrane of the pyloric area and produce in this membrane a substance called gastric *hormone* or gastric *secretin* that circulates in the blood stream, and when it reaches the gastric glands provokes the secretion. The formation of this secretory stimulant seems to be limited to the pyloric area. A similar hormone is formed in the intestine, causing pancreatic and biliary secretion. I cannot say that the discovery of this stimulus to secretion has given any suggestions as to the treatment of stomach diseases. Physiologists, however, have become much interested in this essentially chemical relation of one organ or function to another and are inclined to

think that many of the so-called reflex interaction and co-relation of certain body functions will be ultimately explained on a chemical rather than a nervous basis.

But recent work on stomach physiology has not been limited to studies in its secretion. The muscular movements of the organ depending on its anatomy and the function of its different parts have come in for some interesting investigation. It has been known for a hundred years that the appearance of food in the pyloric area was different from that in the fundus, and that the intra-gastric pressure and muscular activity of the pylorus was greater than that of the fundus has also been known for a long time. Anatomically, there is known to be a difference not only in the glandular formation, but also in the musculature of these two portions of the organ—designating in a general way the right fourth as the pyloric portion, and the remainder the fundus. But it is only very recently that the functions of the two portions have begun to be really understood. Much of the credit for clearing up this field is due to Cannon and his associates at Harvard University. Cannon, in collaboration with physiologists and surgeons, has done a large amount of work on the gastric and intestinal functions and his findings are quoted freely all over the world.

The gastric movements have been studied by Cannon by feeding large amounts of bismuth with the food, and then studying the shadow with a fluoroscope. He finds that the fundus of the stomach is in effect an adaptive reservoir for the reception of food; that the muscular wall of this portion keeps up a constant tonic pressure upon the ingested food mass; that at times it squeezes a portion from the surface of the mass into the pyloric portion, but that so-called peristaltic waves are not observed; that when a fresh bolus of food is swallowed it seeks the center of the mass; that as a result of the comparatively undisturbed condition of the food after the end of the meal, and its not mixing with gastric juice to any extent, starch digestion by virtue of the ptyalin of saliva keeps up for a long time. And here is furnished an additional demand for thorough mastication—namely, the better action of the ptyalin by its intimate mixing with the food.

In the pyloric portion, pyloric mill, it has been called—different doings are going on. Here it is we have vigorous peristaltic effort, by which food which has reached it is squeezed towards the pylorus only to find it closed and then to be squirted back towards the fundus and then picked up and churned back and forth. During this process the food is thoroughly mixed with the gastric juice; proteid digestion is inaugurated and carried to a greater or less extent. Finally the pylorus relaxes and the partly digested food is passed into the duodenum. The vigorous action of the pyloric portion can make up to some extent for incomplete mastication, and it is thought that the greater frequency of ulcer and cancer in this portion is the direct result of traumatism, which such vigorous work necessarily subjects it to. In man it appears that the passing forward and squirting backward of the food occurs in waves about 20 seconds apart, and these can be heard by auscultation. It may be that this can be utilized in differentiating the stomach retention of deficient peristalsis from that of pyloric obstruction—this, of course, before the vigorous waves of the latter condition are visible to the eye.

A natural query is, What is the stimulus on the stomach side to the relaxation of the pylorus? In all probability the pyloric closure and opening is the result of the complex stimuli arising both from the stomach and intestine. From the stomach side, Cannon teaches that it is the presence of free Hcl that calls for pyloric relaxation; whereas the closure is prompt when acid material reaches the duodenum—a kind of protective measure to lessen the influx of acid chyme into the bowel. When the acid mass in the duodenum is neu-

tralized there is probably in the normal digestive tract no further interference with the opening if the free Hcl is present in the stomach. However, Cannon, Abbe and others have proven that after resection of the bowel the pylorus will not relax even under vigorous pyloric peristalsis for several hours; indeed, interestingly near the time Abbe sets for a rather strong agglutination of the stitched bowel.

By means of his bismuth shadows, Cannon was able to study with ease the intervals after ingestion of different types of food before they began to leave the stomach. His observations disclose just about what we should expect, bearing in mind the influences that provoke Hcl secretion and the varying Hcl needs of the various foods. Carbohydrate was found to begin to leave the stomach in ten minutes and to progress rapidly. This kind of food calls for very little Hcl, though it stimulates its flow to some extent, and as free Hcl is present from the beginning, such food rapidly passes to the duodenum, its normal place for digestion. It is said to be a custom with actors and public speakers to take chiefly carbohydrate before appearing on the stage. Nitrogenous food, especially some meats, stimulates Hcl freely, but it also requires much Hcl for combination; for this reason the presence of free Hcl is delayed, and this kind of food leaves the stomach later than a meal of carbohydrate. The stomach is the natural place for meat digestion.

Fatty foods, inhibiting to a great extent the gastric secretion, there is required some time for the presence of free Hcl and fats were observed to leave the stomach very slowly.

These observations can certainly be utilized in practical feeding—especially in cases when it may be well to give the stomach muscles a rest, or at least not to over-burden it.

In conclusion, I may say that the old-time clinical observations that emotion retarded digestion, has had experimental confirmation. Cannon observed that a dog worried by a cat while eating gradually stopped secreting gastric juice.

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GOITERS OF THE YOUNG.

BY C. H. MAYO, A. M., M. D., ROCHESTER, MINNESOTA

One of the most interesting phases of the study of the function of the thyroid gland is its relation to the uterus and sexual pelvic organs in the female.

The thyroid of the mother usually enlarges to some extent during the latter part of pregnancy, although excessive activity of the gland can occur with but little enlargement. It rarely occurs that women become pregnant during the period of an excess of secretion, hyperthyroidism, or during the opposite condition, myxedema or hypothyroidism.

Should the thyroid function be deficient in the infant at birth, the child fails to develop properly, either mentally or physically, and it is in such cases that occasionally marvelous results are obtained by feeding thyroid.

In the human embryo the ductless glands of the neck show very early. The thyroid gland develops from three buds, the greater portion being formed from the median one, which is situated between the lobes of the tongue, and from which the isthmus and greater portion of the lateral lobes are developed.

Fig. I. Case No. 21494. Cretin. Age 16—size of seven-year-old.

The lower poles are formed from buds originating in the fourth branchial groove. It is by anomalies of development that the median bud remains in the tongue as a lingual thyroid, or that accessory thyroids are formed

in these areas of development, and occasionally one or possibly two portions fail to unite with the others as they should at the seventh week of the embryo. Such a condition is common to the vertebrates, but unusual in the human.

The thyroid gland varies in size and weight in the healthy adult from one to one and a half ounces. It is inclosed in a capsule which divides behind into three layers, one of which passes behind the esophagus, one between the esophagus and trachea and one encloses the posterior portion of the gland, fixing the structure to the trachea and the thyroid and croicoid cartilages.

This capsule enclosing so many important structures as well as the gland, is the cause of many of the symptoms of goiter other than those of over-secretion.

In the wonderful provision for a blood supply the thyroid bears out the present knowledge of the importance of this organ. The loss of both common carotids would still leave the inferior thyroids, while in many cases a fifth artery exists in the center below, arising from the arch of the aorta or from one of the common carotids.

At present much attention is being paid to the para-thyroids in studying their function, and, in the surgical operations upon the neck, it is imperative to see that they are not injured. It would appear from the irregularity of their location, their number and their blood supply, that nature had been extremely careless with these glands, or that we are mistaken in their importance.

The cretin may have no apparent thyroid, or may have a very large goiter. Operations upon these tumors are indicated to remove a nuisance, and at times it becomes necessary

Fig II. Case No. 23184. Colloid Adenoma. Age 19.

to relieve dysponea, but operation should not be expected to otherwise improve the condition of cretinism.

If the thyroid is enlarged in these cases it is usually of the fetal type; no lumen in the cells, no secretion, and plenty of connective tissue, or, with more cells, less connective tissue, and cystic degeneration of the whole. In normal thyroid the cells have matured and lined the vesicles, and opened a central space between them for secretion.

Simple or colloid goiter, or diffused adenoma is that condition in which the central space between the cells is greatly enlarged and retains a stainable colloid. If the colloid is granular with recent degeneration of cells, we may have symptoms of hyperthyroidism, the type of exophthalmic goiter which develops in those with goiter of some years' duration. Should flattening of the cells occur in those of the colloid type, there is a tendency toward myxedema.

Encapsulated adenoma may be seen, or rounded tumors occupying a part or the whole of the lobe. Often, by growth within the true capsule of the gland, these tumors develop to a great size and undergo partial or complete cystic degeneration. Such tumors are best ennucleated.

Fig. III. Case No. 24394. Exophthalmic goiter without eye symptoms. Age 18.

The usual exophthalmic goiter shows a wonderful hypertrophy of cells with less colloid, at least in the active areas. Excluding infection and malignancy all goiters come under one of these types.

The simple enlargement of the gland in girls at puberty is a fairly normal condition and indicates that menstruation is becoming established.

In some cases there may be signs of hyper-

secretion at irregular intervals, occasionally amounting to mild exophthalmic goiter without proptosis. These cases seldom require any active medication or special treatment other than hygenic, as most of them recover within a period of months or a few years at most.

It is but rarely that colloid or diffuse adenomatous goiter in the young become so resistant to treatment, or so annoying to pressure symptoms that extirpation or resection of a portion of the gland is necessary, although it may be advisable. At times, however, during this period, an encapsulated adenoma may develop into a rounded tumor occupying some part of the gland. Such tumors are best removed.

Most physicians have a remedy for the cure of such goiters, and these remedies run from amber beads up, externally, with some form of iodine internally. Others lay great stress upon a special diet. We prefer some form of iodine internally with intermittant external irritation over the gland.

Exophthalmic goiter in the young is a most serious condition. It may appear at a very early age; we have had a marked case beginning at the age of five years, and operated upon at seven, with great benefit.

The usual age in which hyperthyroidism develops in the young, is between sixteen and nineteen years. In such cases some form of medical treatment should be instituted to improve the condition, but should not be persisted in when ineffectual, until surgical treatment must assume the burden of a mortality which, from the delay and consequent terminal degenerations, should be in part medical.

SOME REMOTE SYMPTOMS AND EFFECTS OF CHOLECYSTITIS.

BY A. MORGAN CARTLEDGE., M.D., LOUISVILLE, KY.

I have been greatly impressed for many years with the pathological changes occurring in the various tissues and organs of the body as the result of chronic inflammatory disease of the gall bladder or ducts. As the result of much clinical work and many observations, the diagnosis of gall bladder disease has been greatly elucidated, yet, I feel that there is much to be learned along diagnostic lines by a closer study of the remote effects of disease in the gall bladder and ducts.

It will be the object of this communication to call attention to the remote effects of cholecystitis, and the tremendous bearing this disease has as a causative factor in destructive disease of other and more important organs. There is a great paucity of literature upon this subject, and, like many other phases of modern pathology remained for the modern abdominal surgeon to clear up and establish as the result of his daily work and observations. It was my fortune to become identified with a not inconsiderable number of gall bladder cases early in my professional work, and have been observing with the greatest interest the effects of gall bladder disease upon the heart and kidneys, especially the latter. Years ago I was impressed by the fact that practically all the gall stone cases gave albumen reactions in the urine and more or less casts. This was a source of great anxiety to me, until I learned that the kidney changes, promptly disappeared after operation in all except the more aggravated or very long-standing cases.

As early as 1896, I observed and called attention in a paper read before one of our local societies to the great prevalence of cardiac changes in cholecystitis. These at the time were considered the result of bile circulating in the blood and the functional manifestations so common as the result of a sympathetic reflex through the pneumogastric. I now have every reason to believe that most of the cardiac symptoms and changes found in gall bladder disease is secondary to the renal changes produced by the elimination of irritating substances in biliary infections. However, I am not sure that all the changes in the heart are caused in this way. The number of endocardial changes in chronic biliary infection are too pronounced to be coincidental, and the long-continued, though slight, amount of bile circulating in the blood undergoes such chemic changes as to produce endocardial proliferations and finally interference with consistent valve action.

There is no literature directly on the subject as far as I have been able to learn. The following points are in the nature of side lights and may be interpreted as bearing in some way on the subject. I am sorry that the time is so short that we cannot make some laboratory experiments upon the subject.

ANALOGIES.

(*a*) Nephritis is a constant complication of diphtheria. It has been experimentally produced in guinea pigs by inoculating them with the toxines of diphtheria. (Osler, Infectious Diseases, page 414.)

(*b*) Nephritis occurs in acute rheumatism. The diplococcus is freely excreted in the urine of persons suffering with acute rheumatic pericarditis. Nephritis may be produced artificially in rabbits and cats by the inoculation with the diplococcus, as observed by Pain, Osler and Vernon Shaw. (See Osler, page 701.)

(*c*) Kidney complications occurred in six, in a series of 1674 cases of erysipelas.

(*d*) Typhoid fever, typhus fever, smallpox, and lobar pneumonia.

From these facts it is not unreasonable to

suppose that the same complications may follow cholecystitis.

The Role of the Bile.

Composition of the Bile:

Water	91.68
Mucous and pigment	.129
Glycolate of sodium	3.03
Taurocholate of sodium	.87
Soaps	1.39
Fat	.73
Lecithin	.53
Cholesterin	.35

Bile absorbed by the Lymphatics gets into the circulation where it undergoes uncertain chemical changes, which result in the formation of tox-albumins. The symptomatic effect of this is to produce slow pulse, slow respiration, low temperature, irritation of the skin, lassitude, headache and coma. Is it then possible for the delicate renal mechanism to escape?

When the liver is altered, the extractive matters accumulate in the blood. Biliary albumens may produce the following effects:

(*a*) Alteration of fixed kidney cells.
(*b*) Reflex vascular disturbances.
(*c*) Exudation and diapedesis.

Microbic poisons are albumenoid while circulating in the blood, but in the urine they are alkaloid. May it not be the function of the kidney under these conditions to break up the complex albumenoid molecules and convert them into alkaloids? Now should an organic radicle be replaced by an H atom, a poison is produced. (Roger, page 133.)

The chemical composition of bilirubin is C-16, H-18, N-2, O-3. Bilifuchsin is: C-16, H-20, O-4. Biliprasin is: C-16, H-22, N-2, O-6. These organic radicles combined with Ca form biliary calculi, but if one of these radicles should be replaced with an H atom, a poison is formed. Example: Nascent H converts bilirubin into C-32, H-44, N-4, O-7 (Orobilin), or in the feces, stercobilin. Taurocholic acid is an example of the substitution of an organic radicle by an H element: H^2 H^4 SO^2 C^2 (Bartlett, Organic Chemistry).

Musser says that albumen can almost always be found in the urine in cases of infected gall bladder.

Cases of acute infectious icterus of a benign form have long been recognized and epidemics described by French observers, and by Weiss in '66.

In 1886 Weil emphasized the acute onset, extremem development of jaundice, tender and enlarged spleen and accompanying nephritis.

Jaeger described a baccillus of the proteus group as the pathogenic factor in a garrison epidemic at Ulm, in 1892. He named it the bacillus proteus fluorescens.

"In infectious icterus the kidneys show a diffuse tubular nephritis."—Weil. "The albuminuria is usually of a considerable grade and hyaline and granular casts are present in large numbers. The urine also contains bile pigments and sometimes red blood cells." —Weil. "The nephritis gradually clears up." The tendency to hemorrhage from the various organs and mucous membranes of the body has long been noted as a result of long existing icterus. This is evidently twofold in its nature, occurring from the operative wound as it sometimes does, the element of mucus admixture which retards coagulation, is a factor. But the altered blood vessels and circulatory stasis is no doubt the chiefest cause. Short of pronounced jaundice there is a tendency to bleed freely from mucous membranes in chronic catarrhal disease of the gall bladder. I have been able to establish a clear relationship between gall stones and menorrhagia where a thorough examination fails to reveal a local source of an existing menorrhagia, I strongly suspect a lesion of the gall bladder, probably calculi.

Very slight, I may say imperceptible, increase in the absorption of bile will increase the normal menstrual flow. In my experience it requires rather aggravated icterus to give rise to kidney hemorrhage. In the aggregate, symptoms caused by the circulation of bile

in the blood we have the clinical picture of so-called biliousness of the ancient authors, and of the laity today, viz, headache, drowsiness, fatigue, furred tongue, neuralgia, cold feet and constipation. This combination of symptoms really toxic in character, has the greatest significance for the surgeon or internist. What is biliousness, so-called? Simply the retention in the blood of toxic substances of varied origin. It has no connection with the liver proper, but it is an evidence of trouble in the biliary passages or gall bladder, viz: absorption of bile from impeded outlet, this absorbed bile in turn irritating the kidneys which interferes with the elimination of important toxic principles from the blood. These toxic principles being the ordinary ones eliminated by normal kidneys, plus those added by the bile, directly and indirectly, for indirectly we have many stercoraceous ferments formed, the absorption of which still further hampers the kidneys. Where the ancient portion of calomel relieves in part this condition, it does so by removing part of the intestinal ferments and indirectly lessening the burdened kidney, also directly by increasing the kidney action. The logical deduction from the premise, which practical experience abundantly proves is that recurrent biliousness, so-called, is a positive indication of gall bladder disease, most likely extending to the ducts.

What we want for a time is less about cholelithiasis and more about chronic cholecystitis and cholangitis; these are, in my judgment, as numerous as where calculi form, and between the two, what a startling chapter figure it makes in human pathology! I dislike to appear to speak immodestly, but truthfully I do not believe the half has been told. Fourteen years and one month ago it was my privilege to state, at the meeting of this society, in this city, that, "We should consider any appendix once so affected as to deserve the name of appendicitis whether from tubercle or trauma (enterolith), a lastingly diseased structure; and fancied cures, but quiescent state the result of very easily recognized conditions." These views were considered too radical that day. I venture to say the same of inflammatory disease of the gall bladder and ducts today. If I can be cured of infections of the gall bladder and calculi by medicinal agents, I don't want to be cured that way. I want the gall bladder and ducts drained so as to get a lasting cure. I would wish to avoid cardiac and renal changes which are inevitable and finally fatal.

The internist should take a large pencil and score down syphilis, alcohol and rheumatism as causes of Bright's disease, and raise chronic infections of the gall bladder from cholelithiasis and other causes to first place.

The recurring bilious, and families with inherited liver disease, headache and malaria, should have a simple cholecystotomy and drainage. Likewise the dejected and melancholic. In addition to the usual symptoms of gall bladder disease many valuable diagnostic signs can be found in the enormous category of remote symptoms and effects.

WHAT WE SHOULD KNOW ABOUT MALARIAL DISEASE.

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Malarial disease is an infection* of the red blood corpuscles with one or more species of a hematozoon officially known as the *plasmodium.* Three species are known: *p. vivax,* which causes the "benign" *tertian* fever with its modifications; *p. malariae,* which. maturing in 72 hours, produces (when in single broods) *quartan* paroxysms (plasmodium malariae is the parasite of *quartan fever only* and the name should not be in a generic sense); *p. precox* occasions the various types of fever grouped under the head of *cstivo-autumnal* by Marchiafava and Celli (*piccoli plasmodi*). Canalis as early as 1889 called attention to two types, one maturing in 24 hours, the other in 36-48 hours. Grassi and Feletti, and Mannaberg, and, later, in this country, Craig, insist upon a differentiation of species for these types, but it is more likely that they are merely differences in strain or environment. For instance, the *p. immaculatum* segments before it has reached half the size of the containing red blood corpuscle, resulting in at most 6 or 8 segments, and before an appreciable amount of melanin could have been metabolized; its crescents, developing more slowly, always have the characteristic ring of pigment. The pigmented quotidian *p. precox* is so generally associated with the unpigmented that it would be more than a coincidence to have this double infection so constant. The *malignant, tropical* or *estivo-autumnal tertian, Laverania* (order of crescents, Grassi and Feletti), matures in about 48 hours, but the broods group so diffusely that clinically it has been recognized as the parasite of the irregular fevers. The clinical types are the malignant tertian, remittent and continued fevers. All estivo-autumnal parasites have crescents as "resting" forms, and the official name of the species for all three is *p. precox.*

CLINICAL TYPES. Single groups of *vivax* produce fever paroxysms lasting approximately 11 hours, *according to the temperature chart, not the statement of the patient,* about every 48 hours, usually beginning in the forenoon. Thus, a chill may come at 10 a. m. on Monday and another at from 9 to 11 on Wednesday. Two groups, maturing on alternate days, give us quotidian paroxysms, and we say we have *quotidian tertian,* or *double tertian;* the time of appearance of the alternate paroxysms need not correspond to the time of the primary group. Thus, we may have a chill at 10 a. m. on Monday, one at 1 p. m. on Tuesday, one at 10 on Wednesday, one at 1 p. m. on Thursday, etc. If the two groups are only half a day apart we can have two paroxysms on Monday, none on Tuesday, two on Wednesday, etc. This is *double quotidian tertian. Triple tertian* gives us two paroxysms Monday, one on Tuesday, etc. Two or three cases of *quadruple tertian* are on record, but it is more usual for multiple groups to mature diffusely, giving us tertian remittent and continued fever; rarely, we have symptoms of perniciousness due to this parasite. The word tertian is used both for periodicity and species; hence it should be qualified, e. g., benign tertian, malignant or tropical tertian; the former is due to the tertian parasite (*p. vivax*), the latter to the estivo-autumnal parasite (*p. precox, var. Laverania*).

Single groups of the *quartan* parasite (*p. malariae*) produce 10-11 hour paroxysms about every 72 hours. Two groups give, for

* For animal parasites the term "infestation" should be used. It is as yet not generally applied to blood parasites, only to intestinal.

example, a paroxysm on Monday, one on Tuesday, none on Wednesday, one each on Thursday and Friday. This is *double* quartan. Three groups fill in all the days, hence we speak of it as *quotidian* quartan; it is most generally designated as *triple* quartan. Infection with the quartan parasite may give rise to indefinite clinical types. It rarely goes into the resting stage and may persist for years. Quartan infection is most difficult to eradicate, although yielding readily (symptomatically) to small doses of quinine. If in double quartan we have the history only of the paroxysms on Tuesday and Thursday we may think we are dealing with the tertian parasite—or if we see the case on Friday we may be deceived into believing that we are dealing with a tertian which has just become "double"; or, the alternate group may have only just become massive enough to begin to show on Thursday. Thus, in a variety of ways we can be deceived by a quartan infection. The diagnosis is important because it calls for more persistent treatment; it can only be made by examining the blood. I have a number of times fallen heir to cases of quartan infection because the first physician allowed too many relapses to occur.

To recapitulate, the estivo-autumnal parasite gives us quotidian paroxysms by maturing precociously; double malignant tertian cannot cause quotidian fever, because the duration of the paroxysm is so long that two groups give us remittent fever, the rise due to the second group intercepting the fall of the first. The quotidian estivo-autumnal fever, then, is due to single groups of parasites having a 24-hour cycle; quotidian tertian is due to two groups of the benign tertian parasite (having a 48-hour cycle) maturing on alternate days; quotidian quartan fever is due to three groups of (72-hour cycle) quartan parasites, maturing 24 hours (or nearly) apart. Tertian fevers, as we have seen, may be caused by any of the three species, typical tertian intermittents being in the vast majority of cases due to p. vivax. Malignant tertian, from the p. precox (48-hour type) is characterized by a longer duration of the fever, a lower and broken fever apex, and rarely exists in the pure form, since the groups tend to brood diffusely, some maturing in 36 hours, others not under 50 hours; this finds clinical expression in remittent fever, with perhaps an indistinct second and rarely a third chill; it is most often confounded with typhoid fever. The tongue, the facies, the belly, the complexion, the leaky skin at times, the restlessness and pains, ought to put the clinician on his guard and not let his malarial patient get into a "typhoid" state or lapse into perniciousness. In either case there is apt to be diarrhea, gurgling, or even hemorrhage. Pernicious fever may come from all three species. Thus we see that, except when we have frank, single-group intermittents, we cannot undertake to differentiate species from clinical symptoms.

A few words as to perniciousness. This is most often due to 24-hour types of estivo-autumnal parasites. In the peripheral blood these appear only as small, very delicate, symmetric rings. Others of the brood may have become crescents; extremely rarely we may find segmenting bodies, from which we can differentiate types; six to eight segments speak for the quotidian, whilst malignant tertian may divide into as many as thirty (I have one preparation showing 28 segments). Brood-containing corpuscles are apt to become impacted in capillary vessels, producing emboli, which

Figure I.

in turn filter back great numbers in localized areas, nearly always in certain organs or groups of organs. According to localization we have cerebral (comatose, etc.), pulmonic, gastralgic, choleraic, neuritic, diaphoretic and other types of pernicious fever. Hemorrhage is but an incident and does not justify a separate type. Likewise, hemoglobinuria is due to death of plasmodia en masse with liberation of endocellular hemolysin, is a remote event, does not attack new-comers, is not necessarily embolic, and has no place in the group of perniciosae. Pernicious fever usually develops gradually but it is not very rare to have a pernicious first paroxysm.

Latent and larvate malaria. Since the introduction of precision in blood examination one hears very little of larvate malaria. Cases of periodic neuralgia, urticaria, and a great variety of periodic symptom-groups are now probably less often ascribed to malarial infection than is really the case. Those who examine blood probably err on the other side, whilst those who are in the habit of treating malaria symptomatically may treat frontal sinusitis, etc., for malarial neuralgia. As to latent malaria, it is unfortunately still the practice to ascribe almost every kind of chronic ailment to "a touch of malaria." I have had cases referred to me as "malaria in the system" that had reached the point of incurability and whose real condition should have been long ago recognized, viz: visceral malignancy, gall bladder disease, abscess of the liver, miliary tuberculosis, latent sepsis, dysentery, uncinariasis, Banti's disease, malignant endocarditis, syphilitic fever, etc.

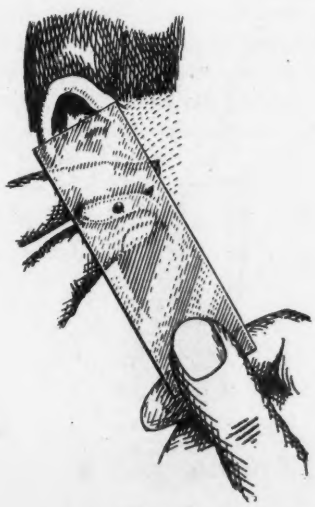

Figure II.

Persons who have been residents of malarious districts may acquire a relative immunity and harbor malaria parasites without having symptoms or at least without having fever. It is quite common for such persons to have "resting bodies," which are the gamete or sexual forms of the beginning of the long or mosquito cycle. Only persons harboring this phase of the organism can transmit the infection to mosquitoes, and this is most often the case with those up and walking about. Such persons are a menace to the community, and they may infect mosquitoes in their own environment, and become re-inoculated with active malaria. Such an event occurring in a refugee into anopheles territory may wonder where his fever came from, since he fancies he has just run away from it, yet he has actually started a focus. This responsibility of a malaria-infected individual should be explained to him, and the physician himself should never forget to be alive to his duty in such cases and insist upon having the patient remain under treatment. This is the more important since resting bodies are absolutely refractory to quinine and their appearance must be prevented.

Diagnosis. This is said to be accomplished by the clinical symptoms, the temperature chart, and the examination of the blood. For ideal work it is not only necessary to recognize the parasites but also the species, since the treatment and prognosis then become more precise. We have already alluded to the fallacies of the clinical symptoms. The therapeutic test, especially in cases of sepsis, is

worse than useless. Latent malaria may become active when some condition lowering resistance, such as shock, bad hygiene, injury, the puerperium, and surgical operations, intervenes. This is the source of our drinking water malaria, watermelon malaria, etc. Now, if a puerpera has a chill, followed by fever, and quinine is given, either its oxytocic power or accident may cause the expulsion of a septic clot, and a case of "malaria" has been cured. The matter is more complex if a pus focus evacuates after a dose of quinine and the fever falls. Here the fancied security of having aborted an attack of malaria may lead to serious complications. We see it stated as axiomatic that leucocytosis is positive evidence against malaria. This is wrong. Malarial leucocytosis as a rule is low but exceptionally, especially the vaso-motor kind of pernicious and hemoglobinuric forms, may be very high. The *relative* increase of large mononuclears here becomes diagnostic in case of failure to find the parasites. In the presence of a leucocytosis, the increase in large mononuclears only becomes apparent if we make both absolute and differential counts, and then calculate the absolute number of each variety. It is then possible to tell malarial variation even if only four or five per cent. of the leucocytes are mononuclears. Whilst the normal number is about 300 per cm. (which rarely quite doubles during a septic leucocytosis) we find in malaria, in any stage, as many as 800 to 1,000 per cm. Then, too, there is the quinin leucocytosis, coal tar and bath leucocytosis, which is so generally overlooked. The normal fasting blood rarely contains over 6,500 leucocytes. If we take the sample with due regard for the conditions normally influencing leucocyte movement we can get far more information from the study of the leucocytes than is ordinarily the case.

Whilst clinical symptoms and quinin test are not to be relegated entirely, especially by those with no great experience in malaria blood work, it is quite the practice to persist in the exhibition of toxic doses of quinin long after the astute clinician should be aware that he is dealing with some other fever. On the other hand, certain types of malaria are incredibly refractory to quinin—sometimes because the criterion used is "cinchonism," which in some persons develops after very small doses or very little absorption. With the addition of suitable correctives larger doses can be borne.

No one should attempt to examine blood for malaria unless he has studiously practiced some approved method and found it reliable and himself competent. Unfortunately, none of the ready-made stock stains can be depended upon. Many are faulty even before they are sent out, and all of them deteriorate sooner or later, and sometimes quite suddenly. The most important thing to learn about blood stains is to know when a film is improperly stained and to be able to differentiate faults of technic from those of deteriorated stain. All the one-solution stains, whether Goldhorn's, Leishman's, Wright's, Hastings', Giemsa's, etc., tend to lose titer and become acid, only the eosin taking hold. Another mistake commonly made is to use them all alike, whereas each author's method must be strictly followed with his own stain. After considerable experience in teaching students and practitioners I have evolved a method which comes nearer being automatic than any other I have ever read or heard of, and I have tried scores of them. The method

Figure III.

is more suitable for malaria than for leucocytes; even here it is superior for all except basophiles, which have no practical significance anyway. It should be added that neither Ehrlich's nor Jenner's stain have any place in malaria work. Of the one-solution stains I prefer a fresh home-made Goldhorn, Hastings or Wright in the order given; one and one-half grains of the powder is dissolved in an ounce of freshly redistilled methyl alcohol and the solution kept in a cork-stoppered bottle *and never removed except by pouring from the bottle.* Making up more than this is waste, as it will not keep.

THE AUTHOR'S METHOD.

Provide a cork-stoppered homeopathic vial containing absolute alcohol. Drive the butt end of a steel pen firmly into the under surface of the cork and break off one nib of the pen; when the bottle is corked, the pen point becomes sterilized. (Fig. 1.)

Next, have a supply of microscope slides, a 50 cc. bottle of absolute alcohol, a number of pieces of plain gauze, napkin size, as used in throat practice. Now proceed as follows:

1. Wash the hands and rub off with alcohol.

2. Prepare six slides by wiping them first with alcohol, then with dry gauze.

3. Seated on the right of the patient, facing in the opposite direction, grasp the lobule of the patient's right ear between the thumb and index finger of the left hand so as to cause turgescence; rub it briskly with a wisp of gauze dampened—not saturated—with alcohol.

4. Plunge the pen into the presenting end of the lobule with a quick stroke to the depth of 3 mm. (⅛ inch).

5. Squeeze very gently and wipe off the first drop—the next will flow more freely—let it collect in a small, globular, not flat, drop on the surface.

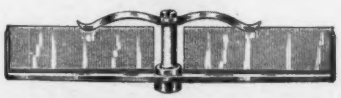

Figure IV.

6. With the right hand pick up one of the slides, lower its distal end on the index finger of your left hand, as shown in the illustration (Fig. 2), then depress the proximal end of the slide until the apex of the drop is just touched, taking on no more than the size of a typewritten "O." Under no circumstances compress or blot the drop.

7. By pronating the right hand, the slide is tilted upside down toward the operator, taken into the left hand, the drop being now on the upper surface of the slide close to the grasping thumb.

8. With the right hand a second slide is now taken up, its lateral edge placed transversely to the surface of the first slide and proximally to the drop, tilted forward at an angle to suit the operator (about 45 degrees) and gently and very slowly swept across the blood, so as to distribute it over the surface, resting a moment *on* the drop to allow the blood to spread laterally.

9. Fan the filmed slide rapidly in the air until dry. The remainder of the slides are filmed in the same way, wiping off the bleeding point and extruding a fresh drop before each smear.

It is best to practice on a willing victim, at first without drawing blood to get the drill, then with blood to learn how to get good spreads. It can also be practiced before a mirror with blood from one's own ear. The mastering of this technic is important because it enables one to send blood to the laboratory if not prepared to examine it. Scrupulous cleanliness, some deftness and speed, absence of dust and air currents and strict adherence to directions are necessary for success.

Flies will quickly eat off dried blood if allowed access.

Smearing with a needle, cigarette paper or by pushing the slide in advance of the blood or by holding it lengthwise or making the film

on cover glasses need only to be mentioned as ways not to do it. The above method leaves all the elements undamaged on one film. It distributes the leucocytes ideally for differential counting; at the lateral fringe they are massed in proper proportion; at the terminal fringe the largest elements are collected, enabling one to judge marked relative increases of large mononuclear cells off-hand. Crescents and segmenting bodies also group here.

STAINING OF THE FILM: I.—EQUIPMENT FOR BLOOD WORK.

1. A table before a window facing north, to eliminate the direct rays of the sun.

2. A sink or washstand close by, over which is a rack for the stains.

3. A standard microscope with triple nosepiece, Abbe condenser, a good 2 mm. immersion lens with wide angle and good penetration, even if at expense of flatness of field, the usual 4 and 16 mm. dry objectives, a one-inch eye piece and mechanical stage.

4. A urine centrifuge with one graduated tube (Fig. 3) and Author's slide drying head which is exchangeable for the tube carrier (Fig. 4).

5. Several 30 cc. pipet bottles (Fig. 5). The ground "stopper" surface should be well paraffined.

6. Slides, coverglasses (square), forceps, test tubes, filter paper, litmus paper, distilled water, etc.

7. Two Pasteur-Petri dishes, 100 mm. diameter.

8. One gram Azur II. Gruebler, dry.

9. Ten grams eosin, water-soluble, yellowish, dry (Gruebler).

10. A one per cent. solution tannin containing a few drops of chloroform.

11. 500 cc. Merck's guaranteed methyl alcohol. Commercial wood alcohol redistilled over caustic potash will answer.

12. 100 cc. C. P. Aceton. This removes precipitates from stained slides and is useful for final washing of blood pipettes.

II.—METHOD.

Make up 25 cc. one per cent. solution of II. in distilled water and 25 cc. aqueous eosin solution, add a few drops chloroform to each and keep in dropping bottles; select two delivering the same number of drops per cc.

1. Flood blood slide with methyl alcohol for one minute, during which time it must be covered by the bottom half of a Petri dish to prevent evaporation. For this purpose it is convenient to have a slip of thick glass, smaller than the slide, cemented on a small square of window glass. The slide is laid on this, the alcohol poured on and the cover placed over it; in this way the draining off of the alcohol is minimized.

2. While the slide is "fixing," measure 7 cc. distilled water in the graduated centrifuge tube, drop in exactly four drops of the eosin solution, shake, drop in six drops of the azure, shake again, and pour into the lid of the Petri dish. Into this place the still wet slide (inverted), letting one end rest on the edge. The staining solution will just cover the blood smear; it is left on for exactly five minutes. Remove, rinse rapidly in a glass of water and immediately dry on the centrifuge.

The slide can now be examined dry under the low power to see if the nuclei of the white cells are stained very deeply. If this is not

Figure V.

the case the amount of blue in the mixture was insufficient or the methyl alcohol was acid. The correct quantity of blue can be judged by examining the mixture in the dish immediately after it is made over a white surface, when the mixture must be quite blue. If the nuclei have been found too pale, the film is stained as before, using one drop less eosin. Old films are apt to come up too blue; this can be reduced after drying by pouring on tannin solution and immediately rinsing off with water, and drying on the centrifuge. Precipitates can be removed by wiping the dry slide with a pledget of cotton dripping wet with pure aceton; it will exaporate at once. This cotton must never be used a second time, because it rapidly absorbs water.

Slides must never be dried with blotting paper or by heat nor allowed to dry spontaneously; in the absence of a centrifuge they must be waved in the air until dry.

A good field is now selected under a low power and a drop of immersion oil placed on it, then the film searched with the 2 mm. objective without a coverglass. If the slide is to be preserved, a coverglass is put on without balsam, using more oil if necessary. (Canada balsam is acid and soon bleaches the film.)

III.—THE APPEARANCE OF THE SPECIMEN.

The red cells are orange to pink, but if slightly grayish, it is a good fault. The lymphocytes have a partridge blue cytoplasm and a purple nucleus. The neutrophiles will be pale pink or heliotrope, showing purple neutrophile granules, the nuclei deep violet. The large mononuclear cells have a pale blue cytoplasm, a rather light-colored nucleus and a few deep purple perinuclear granules. The platelettes are heliotrope with deep purple nuclei; often one or more are found within a red cell and *mistaken for malaria parasites.* These latter *are never any color but pale blue,* the denser portions deep sky-blue; their chromatin is *carmin-red* and is the only element taking this color. By this means they can be positively identified. Unless the normal elements take on the colors specified we can not be sure of having stained the plasmodia. Just before the paroxysm and in the early hours of the fever it may take an hour to find a single one. After twelve hours they become quite numerous and are usually easily found. Quartans are found in every stage; estivo-autumnals brood diffusely and small rings are generally found if careful search is made, although most numerous during the remission.

IV.—APPEARANCE OF THE PLASMODIA.

The youngest forms of the parasite are differentiated by the beginner with difficulty. They appear of pale blue rings one-fourth the size of the containing cell and in the beginning may be at the edge; they enclose an achromatic zone, at one pole of which a minute dot of a carmin-red color can be seen. The quartan is the most compact and dense of the three, the estivo-autumnal the most delicate and pale. In the latter it soon divides and spreads around the ring, forming a horseshoe; this is absolutely characteristic. The tertian rapidly becomes varicose and filamentous; if the film has been dried rapidly, the most delicate lace-work results; at least one pigment granule appears within the thickened blue cytoplasm before the chromatin divides, which it does completely, into round granules, except when very filamentous. The quartan is deep blue in all stages, attains no growth under 18 hours, shows black, coarse pigment early, tends to spread across the cell in a broad ribbon, the chromatin divides late and slowly. The estivo-autumnal parasite retains its slender ring shape until it is quite large, forms pigment late and only slightly if at all, and which clumps early into one large block. The segmenting tertian makes over sixteen segments in a double row, the quartan eight to twelve in a single row, its pigment being now quite abundant, coarse and black, that of the tertian being more brown owing to its fineness and transparency.

The resting bodies can be differentiated early but the consideration of these would carry us too far; those of the benign species are spherical, those of the estivo-autumnal are sausage-shaped (usually called crescents). So-called flagella develop only after the blood has been shed for some time; this requires special technic. For ordinary observation they can be seen in fluid blood under a coverglass. The accompanying pictures will explain differences. The subjoined table will give the chief characteristics of the three species in parallel columns.

	P. VIVAX.	P. MALARIAE.	P. PRECOX.
Short cycle	48 hours	72 hours	20 to 50 hours.
Gametes	Spherical	Spherical, smaller, coarser pigment	Crescentic.
Young Parasite	Rather compact ring, achromatic center	Very compact, dark blue ring perfectly white center	Pale, open, delicate ring, hemoglobin shows through.
Chromatin	Does not stain w. methylen blue	Does not stain w. methylen blue	Stains dark blue w. methylen blue.
Chromatin divides	In granules *after* pigment appears. Sometimes forms ribbon	In granules *after several* pigment specks appear	Spreads laterally very early whilst ring is very small.
Pigment	Early, fine brown.	Early, coarse black	Late or none, soon clumps.
Pigment in fresh blood	Fine, actively dancing	Coarse, black, lazy	Very active at first.
Motility in fresh blood	Actively motile, filaments early	Slowly motile, remains compact	Actively motile, retains ring shape.
Appearance of fresh blood	Cell early enlarges and becomes pale	Cell contracts and becomes darker	Crenation, retraction of cytoplasm, brassy color.
Half-grown parasite	Many secondary loops, varicosities, much fine pigment	Very coarse, dark, varicose, ribbon forms, compact black, coarse pigment	Still, ring-shaped, signet form, slightly pigmented.
Color changes in stained blood	Schuffner's dots with Goldhorn's (red stippling)	Cell stains deeper	Basophile degeneration in deeply stained films (blue specks).
Segmenting	16-24 segments in 2 circles	8-12 segments in one circle	Rarely found; 6-30 segments.
Time of appearance	Most abundant after fever; scarce before chill and later	All stages	Generally only during growing stage (rings) but develop diffusely, hence may be found any time.

(TO BE CONTINUED)

THE USE OF COCAINE IN SURGERY.

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Cocaine has such an extended field that it is the object of this paper merely to touch upon some of the more common uses that may arise in the daily work of the general surgeon. Aside from its direct absorption from a raw surface or from a mucous membrane, the application of cocaine may be divided into three different methods. First, the intra-spinal, which consists of the injection of a solution of cocaine under the dura of the spinal cord. This method has been largely used in this country by Morton, of San Francisco, who has very greatly perfected the technic. However, it is somewhat uncertain, as even in skilled hands there are occasional failures to produce anesthesia. The possibilities of injuring the cauda, or of causing a hemorrhage, or introducing sepsis and so starting a meningitis, are things that may very rarely happen, but cannot be entirely overlooked. But the chief objection is that the cocaine must be used in a strong solution, preferably dissolved in the spinal fluid itself. When individuals have a marked idiosyncrasy for cocaine, as occasionally happens, it would be impossible to recover the cocaine injected intra-spinally, and the rapid absorption of the strong solution might readily be fatal.

In the second method, called "regional anesthesia," the cocaine is injected into the trunk of a sensory nerve, and so blocks sensation over the distribution of the nerve. This method is nearly always combined with the third method, which depends upon the injection into the tissue to be operated upon, whereby the nerve terminals are affected. This is, strictly speaking, local anesthesia. In either the regional anesthesia or the local anesthesia, the amount of cocaine can be regulated to suit the needs of the operator, or what has already been injected can be partly recovered by incising the tissue rapidly, if symptoms of poisoning arise. Then, too, in these last methods, the solution can be used in much greater dilution than in the case of spinal anesthesia.

In operating upon the skin a solution not stronger than one-fifth of one per cent. is all that is necessary. In deeper structures this solution may be diluted to one half this strength. The amount of cocaine used should be accurately measured, and should never exceed one grain. Usually a half grain is all that is necessary in most operations, even in such procedures as an operation for the cure of hernia. The most satisfactory results are obtained by freshly prepared solutions. It has been my practice to have cocaine crystals put up in powder, each powder containing one grain. If necessary these powders may be sterilized by dry heat, though if the cocaine crystals are fresh, this is not essential. Warm water is more effective than cold. In an ounce of warm water is dissolved the contents of one of these powders, which makes about one-fifth of one per cent. solution. In injecting the skin, care must be taken to place the solution in the substance of the skin itself, and not in the subcuticular tissue. Each injection should produce a wheal, as in urticaria. If injected beneath the skin all of the nerve filaments are not reached until a large amount of infiltration has been produced. In very sensitive patients it is well to place a drop of carbolic acid at the intended point of injection. This procedure deadens the first prick of the needle. After

a sufficient amount of skin has been anesthetized the solution should be still further diluted, and the subcutaneous tissue injected with a more generous supply of the solution. This should be done before incising the skin, as the loose connective tissue and subcutaneous fat is much easier to infiltrate before the skin has been cut than afterwards. In operations upon an extremity a tourniquet should be first applied and the region of the nerves injected with a weak solution. Amputations of the fingers or toes can be done entirely painlessly by first injecting the skin in the manner suggested, after applying a rubber band as tourniquet, and then infiltrating with a weak solution around the four points of the nerve supply.

The most successful kind of major operations performed under cocaine is the operation for the radical cure of hernia. In a patient suffering from inguinal hernia the disabilities are great. The chief objection to an operation made by most of these patients is the fear of a general anesthetic. In the young and healthy such a fear has no serious foundation, but in elderly people, in those who use alcohol constantly, or in patients with nephritic disease, the danger of a general anesthetic looms up prominently. In these classes of cases the radical cure under cocaine is particularly indicated. I have operated with cocaine more than thirty times for the radical cure of hernia. Two of the patients were seventy years of age, and one of these had a double hernia. At least ten of these patients were over sixty. There was no death and no serious complication after any of these operations. Union took place by first intention in every instance with two exceptions, where a very mild infection occurred along the skin edges, but did not materially interfere with the progress of the case. In all the cases I have been able to follow there has been no recurrence. It is quite likely that the bodily resistance is much decreased by the use of a general anesthetic, and this, together with the straining on the sutures incident to vomiting after ether, predisposes to suppuration to a much greater extent than the infiltration of the tissue with the weak cocaine solution. As Bodine and Mitchell have pointed out, tissues are handled more gently under cocaine than when a general anesthetic is employed, and this, too, favors healing by first intention. Taking away, then, the danger of the general anesthetic and the risk from sepsis, the radical cure of hernia should be without mortality. The technic employed in these cases has been as follows:

The skin is infiltrated with a one-fifth of one per cent. cocaine solution, the incision being a little higher than is usually the case. The subcutaneous tissue is injected with a one-tenth of one per cent. solution. After incising the skin the fascia of the external oblique is split at the outer angle of the incision, and the ilio-inguinal nerve found and injected with a one-fifth of one per cent. solution of cocaine. The ilio-inguinal nerve usually lies below the incision, and the ilio-hypogastric just above it, though the relation of these nerves is not constant, and their accurate detection will require experience in clinical work or dissections upon the cadaver. The incision is then extended down toward the pubis, and the inguinal canal opened. The tissue around the internal ring is infiltrated, and the sac dissected out. Dissection of the sac should be done largely with sharp scissors. Ligation of the sac often causes some pain, but frequently is done without inconvenience. The pain here, however, when it occurs is slight and of short duration. The operation is usually concluded after the method of Ferguson, which consists in suturing the conjoined tendon and the external oblique to Poupart's ligament without transplanting the cord.

Other abdominal operations may be done under cocaine, but they are often of doubtful propriety except in unusual instances, as the nerve supply can not be controlled. In

selecting a patient for operating under cocaine, due regard must be paid to the characteristics of different individuals. A patient who is naturally nervous and excitable will suffer more from mental anguish and the consciousness of knowing that he is being operated upon, than he will from actual physical pain; so it is unwise to select such cases for cocaine anesthesia. On the other hand, it is not necessary for the individual to be a stoic. If he can keep himself moderately under control, and say when he is being hurt so that more cocaine can be used, a satisfactory and painless operation can be done, provided it is of such a character as lends itself to the employment of a local anesthetic.

THE INDICATIONS FOR THE MASTOID OPERATION.

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The relation that the mastoid bears to intracranial structures is so important that I have chosen the above subject as one of interest to present before this body; and, while I may not interest you in discussing it from an otological point of view, I hope it will be worth your while from the view of a general practitioner and surgeon.

More cases of mastoiditis (both acute and chronic) are seen now than formerly. Not because mastoiditis is more common; not because there are more abscesses of the middle ear; not because we are liable to more colds, but more to our ability to recognize the early symptoms of this important disease. Cases of mastoiditis left to themselves, receiving no treatment except perhaps some hot poultices of flaxseed, soon reach the point where vital structures are placed in danger; and while an operation in the great majority of cases may result in a cure, so far as the suppuration is concerned, one of our most delicate senses is made to suffer, and the organ of hearing is greatly impaired, and the patient remains deaf the balance of his life. It is my purpose, therefore, in this paper, to call your attention to the early operative symptoms of mastoiditis, with a view of maintaining a normal organ.

The indications for a mastoid operation are so evident in the vast majority of cases, that with the application of a few tests, no great skill is required to make the diagnosis certain.

It is the general rule, with some exceptions, that mastoiditis occurs secondarily to an acute purulent otitis media. The drum membrane has usually ruptured, and the canal is filled with a muco-purulent or purulent secretion. In the majority of cases of acute purulent otitis media, there is some mastoid involvement, and pain on palpation can be elicited if sought for. This pain may be due to nothing more than an engorgement of the mucous membrane, lining some of the superficial cells of a highly cellular or pneumatic mastoid, or the antrum, or the large cell at the base of the mastoid might be solely infected, giving symptoms of mastoid infection. These symptoms should subside in a day or so with the subsidence of the acute ear symptoms; but if persistent, they then assume the dignity of symptoms of real mastoiditis, and demand more attention.

In determining the diagnosis, first an inquiry into the history of the case should be made, and, where possible, accurate data kept.

In cases of long duration, for instance, for two or three weeks, we would more than likely have a greater bone destruction than in a case of three or four days' existence. The fact that an attack is a second one would also call our attention to pathological changes that could have taken place in the mastoid, and we would likely find sclerosed bone, indicating that pus forming in this particular case under pressure would be more apt to travel inward toward the brain than outwardly.

Inspection would be our first means of examining the case, and at a glance one could determine whether there was a swelling or redness over the mastoid. But the mere presence of edema or redness over the mastoid should not be taken as conclusive evidence that there is mastoid involvement without a careful examination into the case and an inspection of the canal and drum. An acute otitis externa or furuncle of the canal gives frequently

the same picture as that of a sub-periosteal abscess in mastoiditis, and where there is an association of mastoiditis and furuncolosis, the diagnosis is most confusing. Usually, though, traction of the auricle, pressure in the canal with the finger tip or probe, and pressure in front of the tragus is very painful in furuncolosis, and in the absence of an acute otitis media there is likely an absence of a discharge.

An adenitis might cause post-auricular swelling, resembling swelling of mastoiditis; but it is usually superficial and rarely acutely painful.

The swelling of mastoiditis having progressed to the stage of pus formation or not, usually begins in the post auricular fold. It may be of two kinds, viz: a periostitis, with a subsequent infection of the surrounding tissues; or no tissue infection at all, but the spontaneous rupture of the cortex of the mastoid and the escape of the fluid contents subperiosteally. This is usually the case. This type of mastoiditis with marked swelling, the auricle greatly pushed forward, marked fluctuation, and some pain, alarms the general practitioner; often frightens the aurist, and he is impresesd with the idea that the patient is in great danger. As a matter of fact, such a case is in less danger than if the cortex had not ruptured, where the pent of pus under pressure between the two tables of bone is only waiting for the point of least resistance to give way and an intracranial complication results.

I had occasion, a few years ago, in a large New York hospital, to assist in, or operate upon, 271 cases of mastoiditis. Swelling as a symptom in that series did not occur in but 133 cases or about fifty per cent. Of this number of 133 having swelling, 88 or sixty-six per cent., had reached pus formation.

Swelling usually occurs as an early symptom in children and infants, as does also the formation of a subperiosteal abscess, and it occurs as a late manifestation in adults. The statistics in this series that I have collected corroborate this statement. Where a sub-periosteal abscess had formed in an adult it was evidence of a long-standing disease.

Frequently in cases of long standing, we find in the neck beneath the tip attachment of the sterno-mastoid muscle, swelling without fluctuation. It is hard, brawny, and it is only by a deep incision that pus is evacuated. This is called Bezold's mastoiditis, and it is due to the rupture of the inner plate of the mastoid tip in the diagrastic groove, and it usually occurs in cases of several weeks' duration.

Swelling, then, is not the commonest symptom of mastoiditis, by which one not an aurist can make a diagnosis. Some other symptoms more frequently found must be sought, and palpation is that chief sign. The patient's side or profile should face the operator, and while one hand steadies the head to keep him from drawing away, the thumb of the other hand is used to make pressure.

In many acute attacks of otitis media, there is during the first day or so, some pain in the mastoid, usually elicited on deep pressure over the antrum. This character of pain might be compared to the intense neuralgic pains present in the ethmoid cells or the frontal sinus, during an attack of acute coryza. In the acute ear inflammation, under a free myringotomy and proper treatment, unless complications arise, the pain disappears in a few hours to a day after the onset. The pain should not be considered a pronounced symptom of mastoiditis, for cases operated upon during this stage show only an inflamed muco-periosteum, and it is this swollen membrane that causes the pain. If, however, the pain is persistent, lasting five or six days after the onset, one's suspicion should at once be aroused and operative measures entertained.

Gentle but firm pressure must be made, and the points for selection of pressure are the tip, antrum and post tip, and these in the order named will be the areas giving the greatest amount of pain. It would naturally seem that

the presence or absence of mastoid tenderness could be easily detected in any case, but considerable care must be exercised, for in nervous or irritable patients, and in children, the slightest pressure will often cause them to wince or cry. The facial expression must be watched, it being the most valuable means of determining pain, and, at the same time, it is best that the patient's attention be diverted from himself.

Tenderness, then, on palpation is the most positive symptom in the absence of other signs that should guide the general practitioner in making a diagnosis of mastoiditis, but, unfortunately, this, too, is lacking in certain cases, making the diagnosis more interesting and somewhat confusing.

Cases of mastoiditis having enormously thickened cortices might mask all evidences of pain on palpation. Mastoids having few cells might do the same. Cases infected by an organism that has caused a stir among otologists, the streptococcus capsulatus, almost invariably act slowly and treacherously, yet with destruction; causing little discomfort to the patient, except a profuse persistent aural discharge. These cases have little pain on palpation, except, perhaps, in the very acute stages. I would then consider cases infected by the streptococcus capsulatus, operative ones, even in the absence of swelling and pain on palpation, after three to four weeks' duration, provided there was a profuse aural discharge, with or without temperature and pathological fundus or canal conditions.

Temperature, while an important symptom, is often deceiving. In the majority of cases it ranges from 99 to 102. In adults, it often remains normal, but it rarely ever remains so in children. Where a temperature is 103 or above, one must at once suspect some intracranial complications as sinus thrombosis, brain abscess, or meningitis.

The examination of the canal and drum, while an important aid to diagnosis so far as the otologist is concerned, perhaps will convey little information to the general practitioner. The drum shows those appearances found in any acute ear, as a reddened, bulging drum, landmarks destroyed and a perforation through which exudes a muco-purulent or purulent secretion. The canal shows an important sign of mastoiditis in the sagging of the posterosuperior canal wall near its drum insertion. This indicates a suppurative focus in the mastoid bone, and represents a soft tissue infection.

Furunculosis causes a narrowing of the canal, and might be mistaken for this important symptom of mastoiditis. There is pain on pressure with a probe in the canal in furunculosis; but little or none in mastoiditis.

We seldom see non-perforated drums in mastoiditis except in children. The longer a drum remains intact in a case of purulent otitis media, the more likely the mastoid will be involved. In infants and children the cortex has less resistance frequently than the drum, and mastoiditis develops with swelling and abscess formation before the drum ruptures. In short, a well-marked case of mastoiditis *can* exist without a discharge from the external ear. This one case well illustrates an important point in the treatment of infants and children:

An infant ten weeks old, so far as the parents knew, was in good health, and had not fretted or cried any more than usual. The day before admission the mother, on giving it its morning bath, had noticed a slight swelling and redness behind the auricle. The examination showed a small fluctuating swelling, posterior to where the tip should be. The drum appeared reddened, bulging with no perforation. The temperature was 100. A myringotomy was done, with a free gush of thick, yellow pus, containing streptococci. The next morning the swelling had disappeared, the temperature was normal, and there was a free discharge from the canal. At the operation the most destructive mastoid I have yet seen in an infant was found, and besides

a large extradural cerebella abscess, the cells in the zygomatic region, being well developed for one of this age, were thoroughly infected. The patient recovered.

I am an advocate of a bacteriological examination of aural secretions. It is important and helpful in obscure and doubtful cases of mastoiditis. The pneumococcus and the streptococcus are the organisms causing the greatest number of infections, and appear in all of the malignant types. The pneumococcus is less virulent than the streptococcus in the general run of cases, and given two cases of mastoiditis presenting doubtful operative symptoms, one could afford to wait longer for positive signs in the former (pneumococcus) than in the latter (streptococcus). Where the aural discharge shows a staphylococcus or no organism present, one is safe in concluding that mild or no mastoid symptoms will occur. This is not the invariable rule, for I have seen severe cases of complicated mastoiditis from a staphylococcus infection.

Of late much attention has been given to the making of a diagnosis by blood examinations, chiefly by the estimation of the white cells in point of numbers and in a differential count. The reports of the different investigators of large experience (laboratory men) upon this subject have been so conflicting that, as a general rule, it must be considered of no value. Associated with other symptoms, an increase in the percentage of polymorphonuclear leucocytes, about 80 per cent., would indicate a suppurative focus somewhere, but intercurrent affections and other purulent foci must be borne in mind.

Where there is a leucocytosis and a polynuclear increase we usually find other well-marked symptoms present also, such as an elevation in the temperature.

A resume, then, of the indications for the mastoid operation would show that temperature is valuable, but not reliable. It is often normal in adults; excessively high in children. Blood examinations are of doubtful value, except along scientific investigations, which should not be discouraged. Bacteriological examinations of the aural secretions, besides being of aid in doubtful cases, adds to our scientific knowledge of what causes the disease. Aural examinations, exhibiting nothing but the fundus, sagging of the postero-superior canal wall and the character of the discharge, would hardly warrant an operation in the absence of other more prominent symptoms. Swelling, next to the most important positive sign, is only present in about 50 per cent. of the cases, and a good proportion of these cases are in children. Conditions other than mastoiditis can cause swelling. Palpation revealing tenderness, the most valuable and constant of all the aids, is the safest guide for the general practitioner in diagnosing mastoiditis. This tenderness must not be feigned, but real, and tenderness after the fourth or fifth day must be considered the gravest of mastoid symptoms.

It has not been my intention in this paper to go into the different methods of operating, but, with such well-marked symptoms present in children and adults, as narrated in this paper, I would urge prompt and radical surgical interference.

TREATMENT AND PREVENTION OF TUBERCULOSIS BY TUBERCULIN IMMUNIZATION.

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A specific treatment for any infection must have first, some theoretical foundation which shall be based upon at least a little experimental evidence, and secondly, its value must finally depend upon results obtained from the clinical standpoint. The production of artificial immunity to tuberculosis was considered practically impossible of accomplishment because there is scarcely any clinical evidence that one attack protects from another: Certainly, some immunity must, of course, occur, otherwise the spontaneous recoveries as is often the case would be impossible. For the past fifteen years tireless and painstaking experimental work has been carried on with a view to elucidate the possibility of producing an immunity in the lower animals. It seems that the strongest and most lasting immunity that has been attained thus far is by vaccination with attenuated living bacilli, although the germ-free products and killed bacteria also have some value as an immunizing agent.

In attempting to formulate a method of immunization in the tuberculous subject, the study of the mechanism of artificial immunization in animals is imperative. At this juncture it seems pertinent to discuss briefly the different species of the tubercle bacillus, before taking up the immunity question, Robert Koch, in his masterly monograph published in 1884, in which he gave a long list of cultures of the tubercle bacillus isolated from tuberculous tissues in fowls, cattle and man, was unable to detect any difference among them, declaring that in all the cultures, whether taken from miliary tubercles, lupus or perlsucht, the tubercle bacillus behaved exactly the same. Within the last decade our knowledge of the nature of the tubercle bacillus has been greatly enhanced, until at the present time several distinct species are recognized. These are grouped according to their sources into human, bovine, avian and the tubercle bacillus of cold-blooded animals. This last group of bacilli is of comparatively small importance to the warm-blooded animals, since they are unable to grow at temperatures approaching blood heat, thereby precluding any possibility of infection. The avian tubercle bacillus departs quite considerably from either the bovine or human types of bacilli. This is shown by the lack of susceptibility to certain animals, for example, the guinea pig, which is otherwise so sensitive to the inoculation of the mammilian bacilli, shows a marked degree of refractoriness to the avian type. On the other hand, while birds react very slightly, often producing only localized lesions to inoculations of the tubercle bacillus of human origin. Cases have been reported in the literature where the avian bacillus was isolated from the human being, but such cases are excessively rare. Rabinowitsch calls attention to the occasional occurrence of the avian bacillus in cattle, swine, horses and monkeys, etc., but Simon Flexner is of the opinion that they constitute a small source of danger in the spread of tuberculosis among mammals. To Theobald Smith is due the honor of first calling attention to the distinction in type existing between certain bacilli of human and of bovine origin. Later, in 1901, Robt. Koch startled the scientific world with his announcement "that tuberculosis in cattle offers no source of danger to the human being." He still adheres to his original conception as evidenced by the fact that he reiterated in his Nobel prize address, delivered in 1906. "Human tuberculosis and tuberculosis in cattle are so distinct from each other

that the latter is not to be feared as transmissible to man." It has been proved beyond all peradventure of a doubt that such a statement is not correct, but it has accomplished more than anything else towards stimulating researches throughout the civilized world. Theobald Smith characterized the bovine tubercle baccillus as possessing a greater degree of pathogenic power for mammals than the human bacillus. It also differs from the latter by certain peculiarities of cultural and physiological properties, and that it was impossible to transmute one type into the other under artificial conditions of cultivation. Most authorities agree that transformations of avian, bovine and human baccilli into each other is questionable, even if one variety of baccilli was cultivated for a long period of time in the body of an alien species. Repeated experiments show that tuberculous materials and cultures of tubercle bacilli of human origin when injected into cattle failed to produce a marked or a general tuberculous infection, even when the material was inoculated directly into the circulation.

It is not within the scope of this paper to discuss fully the different types of tubercle baccilli and their pathogenicity. With this statement we may take up the subject of immunity.

The animals which have been of special use for tests of immunity are rabbits, cattle, goats and guinea pigs, the latter being so susceptible to the tubercle bacillus that it is well nigh impossible to produce a high grade of immunity in them. Koch made the first important contribution to the subject of immunity in tuberculosis by means of his tuberculin. During several years following many experimenters had varying degrees of success in the production of immunity in the small animals by the use of tuberculin and attenuated cultures of the tubercle bacillus. The most convincing evidence of the favorable results of experimental immunity in tuberculosis was made by Trudeau as early as 1892. He treated tuberculous iritis in the rabbit's eye by subcutaneous injections of tuberculin, which resulted in a retrogression of the lesion and an apparent healing took place, which, however, ultimately relapsed somewhat, though very slowly. He further found that guinea pigs, rabbits, previously injected with dead tubercle bacilli, live, on an average, longer than the control animals when subsequently inoculated. Trudeau went a step further. He protected rabbits from virulent tubercle bacilli by twice injecting them with an attenuated culture of bird tubercle bacilli subcutaneously at intervals of twenty-one generations, then the subsequent infection of the virulent mammalian bacilli was made into the anterior chamber of the eye. Of the protected rabbits, not one developed a tuberculous lesion, while the controls showed, in the course of a few weeks, a general inflammation of the structures of the eye, the inoculation wound became cheesy, followed later by a more or less complete destruction of the eye ball. DeSchweinitz a few years later reported certain experiments which he had made on guinea pigs and cattle. The former were inoculated with cultures of human tubercle bacilli cultivated for twenty generations. Their virulence was of very low grade to these animals, for it served to protect them against the later inoculation with tuberculous material from a cow. Other pigs, unprotected, were inoculated with the same material, became tuberculous and died in about seven weeks. These and other experiments clearly establish the protective influence exerted by the inoculation of the living attenuated bacilli to more lasting and of greater potency than the immunity obtained by the bacterial products.

Trudeau claims he has never been able to thoroughly protect guinea pigs against a virulent tubercle bacillus, nor has he ever succeeded in curing experimental tuberculosis in them. Behring, as well as Koch, claims to have protected guinea pigs against virulent

infection, but the details of their modus operandi are exceedingly meager. The successful immunization or cure of guinea pigs with the living or dead bacilli or their specific products has possibly been accomplished in rare instances. It can be argued in favor of such experiments that a single demonstrated positive result is far better evidence than countless negative ones, when we consider for a moment that the guinea pig is the most susceptible of all animals, that spontaneous recovery in them is unknown.

Behring, in his Nobel prize address, in the year 1901, announced that it was possible and feasible to vaccinate cattle against tuberculosis. Hist first experiments were efforts to immunize them by means of tuberculin, and later by other toxins from the tubercle bacillus; dead bacilli and attenuated cultures of the tubercle bacillus were also employed. His present plan of immunization, which has become a standard one, for it is furnished commercially, is as follows: Young calves, preferably twelve weeks old, are inoculated intravenously with a standard human culture. Three months later an increased quantity of said culture is again injected. Cattle so treated may be regarded as highly immune, as shown by inoculating them with a virulent bovine culture of tubercle bacillus, with no permanent ill effects resulting. But if unprotected animals were inoculated with the same culture, a generalized tuberculosis developed, producing death in a few weeks. Behring speaks of his vaccinated animals as being "Jennerized," and believes that immunity lasts for life. Simon Flexner, in his admirable monograph on immunity in tuberculosis, which was read before the second annual meeting of the National Association for the Study and Prevention of Tuberculosis, calls attention to the fact that McFadyean is entitled to the credit of the discovery equally with Behring of the immunization of cattle from tuberculosis. Neufeld, a pupil of Koch, and Behring, in discussing the priority of this important question, asserts that while working under Koch's direction he found, as early as 1900, that large animals, the donkey and cattle, for instance, could be protected against the inoculation of a culture of virulent tubercle bacilli, which was always fatal to control animals. So it seems that the priority of this important subject is an open one. As a matter of fact, the principle employed is not new in experimental medicine, for it was practiced long years ago by the immortal Pasteur in vaccination against fowl cholera, anthrax, etc. In 1902, Pearson and Gilliland were not only able to confirm the results of the preceding investigators, but were able to carry the principles of the method a step further by endeavoring to bring about an arrest of the disease in cattle already suffering from tuberculosis. Flexner, in discussing this subject, states that "while it is unlikely that such a therapeutic use of vaccination will ever be made in veterinary practice, the facts are of considerable interest, especially in view of the somewhat similar means employed to arrest tuberculosis in man." If all young healthy cattle were vaccinated before tuberculosis sets in, it would seem that this disease could be entirely eradicated. The gain would be almost incalculable to agriculture. Furthermore, it would enlarge the possibilities of producing immunity in the healthy human subject if it was thoroughly demonstrated in the lower animals over a long period of time that such a procedure was entirely harmless. It seems that the most satisfactory and solid immunity obtained in cattle is produced by a living culture of tubercle bacilli of human origin; this, when injected two or three times, according to Behring, renders the animal immune for life. This statement has not been proved, but it is certain that immunity has lasted at least two years against the inoculation of a virulent culture of bovine tubercle bacilli.

I have had little experience in this form of immunity, but have contented myself with the use of the dead tubercle bacilli, killed at

their minimum thermal death point. My results are all that could be desired. I and my assistants have had under experimentation for the last two years two herds of Jersey cattle, twenty in one herd and twenty-eight in the other. Every year, previous to the last two years, at least two or three animals would die from some form of tuberculosis. Before instituting any form of vaccine treatment, we used tuberculin on every animal as a diagnostic test, which resulted in a positive reaction in six, thus showing that six cows had some form of tuberculosis out of the forty-eight, three tuberculous cows coming from each herd. The vaccine used was a virulent culture of tubercle bacilli of bovine origin, heated for one hour at 60 degrees C. The healthy animals were injected with five milligrams of the vaccine subcutaneously as an initial dose, followed one month later by ten milligrams, and three months later by twenty milligrams; the last dose was one hundred milligrams, which was given six months from the first injection. Two years have elapsed without a death from tuberculosis, except three, which belonged to the original six previously diagnosed as tuberculous by the tuberculin test. The three animals that were in the last stages were isolated while the remaining three, not far advanced, were allowed to run with the herd. Two of these have practically been cured (so far as we can determine as to symptoms, temperature, etc.) by the bovine vaccine. In treating the tuberculous cows, the method of Trudeau in treating tuberculous human beings was in a measure adhered to. We began with small doses and ran up to very high ones, injecting every week. Our initial dose was 1-100 of a milligram with double the dose at the next inoculation, and so on after the method of Trudeau. Of the six tuberculous cows, in which the above treatment was taken up by us, just two years ago, three have died, one will possibly succumb to the infection in the course of a month or so, while two are apparently well. We believe that the injection greatly prolonged life in at least three cows, while the two apparent recoveries were cases of a mild type; still we believe that the treatment aided very materially in their recovery.

All of the foregoing experiments by the different investigators unquestionably establish for tuberculosis the principle of artificial immunization, an achievement hitherto considered as impossible. Although this immunity in very susceptible animals may not be as frank and lasting as we would like, still experiments have proven that it undoubtedly prolongs life when a lethal dose of virulent bacilli be administered to the vaccinated animal as compared with a similar dose given to the unprotected one. Again, it has been shown in the larger animals suffering from localized tuberculosis that by a systematic administration of a tubercle vaccine that there is a retardation in the development of the disease, producing reparative changes in the lesions, and if in the earlier stages often a complete recovery results.

We may ask the pertinent question, why should we not expect just as good if not better results in man than has been accomplished in the lower animals? We know that the human being is not as susceptible to this disease as most of the animals experimented on, because the normal resisting power of the body overcomes the invading organisms, and that spontaneous recoveries in man are exceedingly common. These facts offer us some hope in the future. Already Trudeau has given us some very encouraging figures, showing the comparison of the post-discharge mortality between patients treated and those that were untreated with tuberculin at the Saranac Lake Sanitorium. He says that eighteen per cent more of treated incipient cases are living than of the untreated, while twenty-five per cent. more of advanced cases who received tuberculin are living than of those who did not submit themselves to the inoculation of this agent. It would be en-

tirely feasible to administer a series of inoculations of the vaccine to certain individuals who have a tendency to consumption, even though they had not the disease at the present time. Given a child that is listless, with a pallid skin and flabby flesh, a tendency to inflammations of mucous membranes, in other words a vitiated state of the tissues, a tendency to tuberculosis. In such an individual the tuberculin treatment is imperatively indicated.

The Tuberculins:—The term tuberculin is a broad one, which includes quite a number of different preparations. The first used was Koch's original tuberculin "T. O.," called also "old" tuberculin, to distinguish it from his more recent product, "new" tuberculin.

The "old" tuberculin is the concentrated germ-free culture medium (glycerin bouillon), in which the tubercle bacilli were allowed to grow for several weeks.

The Tuberculin (Deny's) Bouillon Filtrate "B. F." is a product rather similar to the "T. O.," but differs from it in that no heat is used in its preparation and that the bacilli are grown in bouillon.

The Tuberculin "R.," "T. R." or "Tuberculin Residue" is prepared by growing a virulent culture of tubercle bacilli. They are dried in the dark in vacuo and then pulverized. The powder thus obtained is suspended in distilled water and centrifugated at very high speed for about thirty minutes. The water is then drawn off and discarded and the bacterial residue is again dried in vacuo as above and treated in the same manner. This is done several times. It is supplied in liquid form, containing two milligrams of the powder to the c. c. of twenty per cent. glycerin solution.

Bacillen Emulsion "B. E." This is Koch's latest product, which he has suggested for active immunization. It consists of an emulsion of the ground-up tubercle bacilli in equal parts of glycerin and normal saline solution. There are many other preparations used, viz: the Antiphisn (Klebs), Hunter's modification of Old Tuberculin, the watery extract of tubercle bacilli (Von Ruck), the "P. T. O." (Spengler) and many others. The action of all these various tuberculins, according to Pottenger, is similar in part, yet different. Some are much safer to use than others and seem to produce better results than others. His experience with the "P. T. O." (Spengler) is very encouraging. This product is made from bovine bacilli. Pottenger claims that "P. T. O." will immunize against the human toxin much more strongly than the human will against the bovine toxin. He further states that its use will raise the agglutinating power of the blood to a much higher degree that the human tuberculins, and that this power of agglutination will last longer. Taking all the tuberculins into consideration it would seem, theoretically, that the Bacillen Emulsion would offer the most suitable agent to use in order to produce the most marked grade of immunity to the various activities of the tubercle bacillus. It contains not only the body substance of the bacteria but the toxins of the tuberculin as well. The old tuberculin produces antitoxic immunity, while the "B. E." produces bactericidal and antitoxic immunity. Professor Theobald Smith, as a result of his recent studies on animals, suggests virulent uncrushed tubercle bacilli, killed at sixty degrees C., as the best vaccine to be tried in attempts at immunizing human beings. Personally, I have used only the "Bacillen Emulsion" for therapeutic administration, and the old tuberculin, as well as the "B. F.," for diagnostic purposes. My results have been so gratifying that I have no reason for changing to other preparations. I have observed that after a change from one make of the "Bacillen Emulsion" to another (especially in some patients, where they seem not to be doing well) one will be surprised sometimes to find them again on the road to health after the measures previously applied seemed to have reached the limit of their efficacy.

The method employed in the administration of the "Bacillen Emulsion" is according to Trudeau's plan. It is not possible to give in detail the plan for the lack of space, but suffice it to state that he begins with extremely small doses, 1-10,000 of a milligram, and gradually increases, so gradually and at such intervals as to carry the patient to full doses with as little disturbance as possible. By such a method individuals can take ten thousand times stronger than the original dose without any effect. Injections are given about twice a week, of course depending upon the symptoms of the patient, for herein lies the secret of success in the treatment. This plan may be called the practical or clinical method in contradistinction to the scientific or laboratory method, as advocated by Wright and his followers. Wright's method of immunization is based essentially on the determination of the index. In a previous communication about a year and a half ago I advocated the administration of tuberculin governed by the opsonic index; certainly such a method is a brilliant contribution to our knowledge of the mechanism of artificial immunization, but it takes much time and work, necessary in determining the index, which makes it impractical of application in the treatment of tuberculosis by such a method. I find myself slowly drifting to the clinical symptomatology, not only by reason of its practicability of administration, but also by the just as good results that are being obtained by this method of administration as compared with that of Wright.

In conclusion I may state:

(1) Experiments on animals demonstrate the possibility of creating immunity to tuberculosis by the use of the tuberculins.

(2) I am of the opinion that the time is not far distant when individuals showing a tuberculous tendency will submit themselves to the repeated injections of some form of tuberculin, as a means of prophylaxis against this disease.

(3) The clinical method in the administration of tuberculin is far preferable to the one advocated by Wright in which the estimation of the opsonic index is essential.

(4) Tuberculin therapy is proving an essential aid in the treatment of tuberculosis, especially as an adjuvant to the hygienic-dietic open air treatment.

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EDITORIAL

INTRODUCTION.

The Southern Medical Journal was born at an opportune time, and we feel confident will find a ready welcome in the medical literature of the South, which has long needed a medium of this kind that the best thoughts of her medical men shall not only be given to the world, but it is essentially the object of this journal to build up and foster medical thought as clothed by environment and such changes as would naturally come from local conditions and climatic influences. It has long been known that even the most common diseases are more or less changed, or present different features in the South, and indeed in certain localities of the South far different from that in higher parts of our country. It is because of these facts, after numerous requests on the part of the profession in the South, that the staff of this Journal has organized itself, regardless of school influences or personal interests in the sincere determination to keep its pages open to the best from whatever ethical source it may come, and to give to the profession of the South, not only this medium for the registering of their own thought, but to stand firm in the great principles of ethical journalism. We beg to assure the profession of the South that no articles of an advertising nature, and certainly no advertisements of a proprietary nature, shall ever pollute the pages of this Journal. We have determined, even if it causes a financial sacrifice, that the pharmaceutical houses of this country shall never gain control of this journal, and can only use its medium of ad-advertising for strictly ethical purposes. We hope also that the profession of the South will avail themselves of this medium, and give to the world the many excellent thoughts and the many brilliant successes which have characterized the medical men of this section of the country since the times of Ephriam McDowell, Marion Sims, Long of Georgia, and many others to whom we can point with pride as pioneers in the profession, and to whom the entire medical world is under lasting obligations. If the young men of the South would take these examples as their criterion, and do more original work, or even give to the world the original work that they are now doing, it would reflect great credit upon their own section of the country, and place the profession of the South on the proper footing with their medical brothers of the North, and if this Journal can be the means of inciting greater labor, more earnestness and more thorough work on the part of its clientele, it will have accomplished the purpose for which it was created.

While its pages are open, as has been said, to all legitimate articles, yet its staff, being mindful of its duty to its readers, will claim the right to return all manuscript that for any reason they may not feel justified in publishing. So that with these few introductory remarks, we enter the field of journalism with clean hands and ethical principles, and by these we hope to receive the endorsement and patronage of the medical man who desires to keep abreast with the times.

SOUTHERN MEDICAL ASSOCIATION.

The second annual meeting of this Association will be convened in Atlanta, November 10-12, under the presidency of Dr. B. L. Wyman, of Birmingham, Ala. While many of the readers of this Journal are perfectly familiar with this new Association and its purposes, there are others who are not acquainted with all the facts. The suggestion that this Asso-

ciation be organized came from the Nashville Academy of Medicine, and through a committee appointed for the purpose, representatives of the professsion in Georgia, Alabama, Florida, Louisiana, Mississippi and Tennessee were called to a meeting in Chattanooga, Tenn., at the time of the meeting of the Tri-State Medical Association, Oct,, 1906, to consider plans for perfecting the larger organization. The work of organization moved smoothly and far more rapidly than had been expected, for the Association was organized at this first meeting on the single condition that the respective State Medical Associations, at their next annual meetings, should approve. Each of the State Associations, in 1907, unanimously approved the steps that had been taken, and thus the organization became a permanent one.

It is the purpose of this Association to be a purely scientific and social body without any legislative function. The chief object of the Association is to give the young men of the six States composing it, the same chance for advancement that was once enjoyed by that class in the American Medical Association before it grew to be such a large body, as it now is. That it is not antagonistic to the A. M. A. in any sense is shown by the fact that, through its first president, Dr. Martin, of Savannah, Ga., the A. M. A., at its 1907 meeting, was asked to adopt it as one of its branches. The encouragement and training given our young men who have come up through their respective county societies and State Associations, into the Southern Medical Association, will prepare them for the best possible work in the sections of the A. M. A. Without this intermediate Association full development for service in the parent body would be almost impossible, except on the part of a very few of the more determined and aggressive ones. The time of the meeting of the Southern Medical Association being in November, cannot interfere in any way with the meetings of the State Associations and of the A. M. A. Only members of the State Associations can become members of the Southern Association. The and the A. M. A. will be the laws governing the Southern Association.

The scientific work of the Southern Association will be done in three sections, officered for the Atlanta meeting as follows:

Section on Medicine: Seal Harris, Chairman, Mobile, Ala.; H. E. Mitchell, Secretary, Birmingham, Ala.

Section on Surgery: W. F. Westmoreland, Chairman, Atlanta, Ga.; J. L. Crook, Secretary, Jackson, Tenn.

Section on Opthalmology: J. T. Herron, Chairman, Jackson, Tenn.; A. B. Harris, Secretary, Birmingham, Ala.

Any reader now a member of the Association, or expecting to become a member, who may wish to present a paper at the Atlanta meeting, should communicate with the officers of the proper section. The programs are fast filling up.

The Birmingham meeting was not largely attended for the one single reason that the railroads gave us no rates; but from a scientific and social point of view the meeting was a grand success. There will be railroad rates for the Atlanta meeting, therefore the attendance will be large. The programs will be full and rich, therefore the meeting will be a most profitable one.

One advertising page in this issue is devoted to the Southern Medical Association, and at the foot of that page will be found a blank which every reader in either of the six States, not already a member, should fill and forward to our energetic and efficient Secretary, Dr. Oscar Dowling, Shreveport, La.

(G. C. S.)

The announcement to the medical profession of the South that The Southern Medical Journal would be published in Nashville no doubt caused many who received this report to ask: "Why should another medical journal be published in the South?" Some probably questioned the possibility of the success of such a

publication, while others wondered along what lines it would be conducted. So we shall briefly set forth the object and purpose of its existence.

There are already many excellent journals published throughout the South, each of which performs its work well. Still we feel that there is a place for this new journal, such as we wish to establish. It is our purpose to publish high-class, original articles and to furnish in the journal such paper as may be used for satisfactory illustrations so that if an author should desire to illustrate an article the Journal will make cuts and use such paper as to enable the author to present his material to the reader in as good form as it can be published. This is a step forward and offers to the writers and investigators of the South an opportunity to use their own journal.

Another feature which we wish to make very strong in the Journal is the Department of Abstracts, in which will appear monthly well selected and well prepared abstracts from the leading foreign and domestic journals.

The advertisements appearing in the columns of this Journal are scrutinized with great care and are passed upon by the entire Medical Staff. Any preparation or instrument not conforming to the ethics of our profession will not be admitted.

Books, pamphlets, etc., will be received and reviewed with the idea of giving their distinctive and valuable characteristics. News items of general interest will be regularly reported. In brief it is the purpose of the founders of the Journal, all of whom are practitioners, to make it a reliable, instructive, ethical guide.

Can it succeed along these lines? We believe it can and will. Encouragement comes to us from every quarter of the South, and we believe that the profession possesses patriotism enough to give this Journal, if it be worthy, its moral and financial support.

As to the financial success of the Journal, it may be said that there will be but little net profit after expenses are paid. The subscription price is put within the reach of all. But there are two other points to which we look for success, except financial, and these are: first, its teaching function, the way it is received, read and used. Second, its capacity, or opportunity which it offers, for developing a new corps of writers in the South.

We wish it to reflect the best medical thought in this entire section and it is our highest aim to make it something of which we shall be proud. We wish to make this Journal the representative Southern journal, which will record as a whole the South's professional progress. With these expectations we ask the hearty support of every practitioner in the Southern States. (J. M. K.)

REPORT OF CASES

A SURGICAL BLUNDER.

BY A. B. COOKE, A.M., M.D., NASHVILLE, TENN.

Happily for the sick and afflicted, success in the practice of medicine is the rule; failure and error the rare exceptions. But mistakes do occur, and, when not too quickly buried, are sometimes recognized and occasionally even admitted by the party responsible for them.

I am one of those who believe that, in our dealings with each other, the mistakes we make and the defeats we meet in our every-day experience should be discussed as freely as our successes. Certainly the lessons to be learned are equally valuable, and usually far more impressive. Personally, I should be glad to see one session of this society, each time it assembles, devoted to an experience meeting in which nothing but blunders should be mentioned. If we would all be as zealous in criticising our own work as we are in criticising that of others, I am convinced that such session would prove the most instructive as well as the most prolonged of the entire meeting.

In order to set a good example, and show the faith that is in me, I shall utilize this opportunity to briefly report a surgical blunder of which I was recently guilty, and from which I learned several valuable lessons.

Case.—Mr. K., aged, about 32, was brought to me from a Middle Tennessee county for relief from acute retention of urine. He had suffered from a urethral stricture for a number of years, and had experienced several similar attacks which required the use of the catheter for their relief. He denied ever having gonorrhœa or other venereal trouble. The following history of this attack was given by his physician:

The patient was just convalescent from a severe attack of double lobar pneumonia, which had left him in a greatly weakened condition. On the night before he was brought to the city his physician had been called to see him and found him suffering greatly with his bladder, having been unable to void more than a few drops of urine at a time for the previous six or eight hours, and that at the expense of intense pain and tenesmus. Hot applications and other local measures were employed without relief. After free incision of the stenosed meatus, the catheter, sounds, and bougies were in turn resorted to, in efforts to pass the stricture, but to no avail. Finally, after several hours of faithful effort, morphine was resorted to for the relief of the pain, which had become intolerable.

When I saw the patient on the following day, the pain, in spite of repeated hypodermics of morphine, was excruciating, and he begged most pitifully for relief. On account of the tenderness and muscular rigidity, examination of the abdomen was not as satisfactory as desired, but the clinical signs seemed to point unmistakably to a distended bladder. The penis was so swollen and painful from the prolonged manipulations of the previous night that it was deemed unwise to attempt further instrumentation at that time. The indications in the emergency seemed very clear for a suprapubic cystotomy and drainage, and this was quickly decided upon. On account of the patient's enfeebled condition, it was desirable, if possible, to avoid general anesthesia, and we determined to attempt the operation under cocaine.

The patient was sent to the hospital and

preparations for the work hurriedly made. Apparently very little pain from the incision was felt and no difficulty encountered except from the rigid abdominal muscles. When the deep fascia was incised a tense, glistening membrane immediately bulged into the wound. Thinking of no other possibility than that this was the bladder wall, it was at once punctured, when several inches of *omentum* unblushingly protruded. Recognizing what had occurred, a general anesthetic was at once administered, the incision lengthened, and the peritoneal opening closed with catgut. This area was then walled off with gauze and the operation proceeded with. After some little searching, the bladder was discovered deep in the pelvis, fully three inches from the anterior abdominal wall. Owing to its deep position it was reached with considerable difficulty, and, on being incised, proved to contain not exceeding *four ounces* of urine. The bladder wall was greatly thickened and contracted and the urine was thick, cloudy and offensive. The patient *had* retention and the bladder *was* distended—but——!

The operation was completed by suturing a drainage tube in the bladder, and the patient suffered no ill consequences of any kind from his experience. Ten days later a general anesthetic was again administered and an entrance per urethram effected, after which the abdominal wound rapidly closed, and the patient left the hospital three weeks from the date of the first operation, practically well, and *exceedingly grateful!*

The following points in this case are worthy of note: First, that the patient had a urethral stricture without a history of urethral inflammation, specific or otherwise; second, that he had a severe case of chronic cystitis which had existed long enough to cause a contracted bladder, and yet had not suffered enough to seek regular treatment.

By way of extenuation (not justification) of the blunder above recorded, the following several points may be mentioned:

First—The length of time the attack had been on and the extreme abdominal tenderness and muscular rigidity which had resulted, thus masking the physical signs and rendering the abdominal examination misleading.

Second—The enfeebled state of the patient's general health, resulting from the pneumonia, which made it desirable to avoid general anesthesia.

Third—As a result of the two points mentioned, the impossibility of retracting the edges of the incision so as to clearly expose the underlying tissues.

Fourth—The fact that the tense peritoneum, closing, resembling a distended bladder, at once presented in the incision. This was probably due to increased intra-abdominal tension, caused in part by muscular contraction and in part by the flatulence resulting from the morphia exhibed during the preceding twelve hours.

Fifth—The haste which seemed to be necessitated by the conditions under which the operation was done.

In conclusion, I would emphasize two facts: First, that the diagnosis was correct—the patient *had* retention and he *had* a distended bladder; second, that the treatment instituted justifies itself to this extent at least—the patient made a speedy recovery, and remains well and happy.

CLINICAL NOTES

CLINICAL NOTES ON FRACTURES.

BY DUNCAN EVE, A.M., M.D.

Nose.—It is important to replace the fragments and make provision for their retention before the end of the first week. A small roll of adhesive plaster applied on each side of the nose and held in place with some pressure by a strip of plaster applied across the rolls from one side to the other may afford all the support that is needed. The application of collodion makes a good dressing in simple fractures, provided it is applied in several layers. If any difficulty is experienced in reducing the fragments by manipulation from without, the reduction may be accomplished by first spraying into the nostril a 4 per cent. solution of cocaine and introducing for a leverage a single blade of a long but strong pair of dressing forceps, rather thin at the end previously protected or padded with a little cotton twisted around the same.

Zygomatic Arch, with Depression.—Matas, of New Orleans, advises the following: The patient being under a general anæsthetic, and all the necessary aseptic precautions having been taken, a long semi-circular Hagedorn needle threaded with silk should be entered one inch above the displaced fragments, passed well into the temporal fossa, and made to come out half an inch below the arch. The silk thread is then utilized in drawing a silver wire around the bone, after which the bone is pulled into place and retained by securing the wire over a firm pad. The wire may be removed in about ten days.

Lower Jaw.—Where the teeth on the opposite sides of the fracture are wired, I have always gotten better results by also wiring both fragments of the lower jaw to the opposing teeth of the upper. It is generally best to trust to the mechanical ingenuity of a dental assistant than to place any dependence upon special appliances to be found in the stock of an instrument dealer.

Clavicle.—In addition to Sayre's dressing, I use a third broad strip of plaster around the elbow and body, with the idea of increasing and maintaining the outward position of the shoulder by correspondingly increasing the power of the lever controlling it.

Humerus (Shaft).—This bone is more liable to non-union than any other long bone. Treves believes that this is due to the interposition of muscular tissue between the fragments, and to the lack of fixation of the shoulder and elbow joints. Hamilton attributes it entirely to the movement induced by leverage of the fore-arm.

The proper plan of treatment is to apply an internal right-angled splint and a long external shoulder-cap; pad both of these well, especially the right-angled splint at that part where the inner condyle is to rest. This splint should reach from the axillary space to the end of the fingers, the fore-arm being fixed at a right angle with the arm. Apply the splint while traction is made on the arm from the elbow, and secure well the arm and fore-arm to the splint by two or three wide strips of adhesive plaster, placing the fore-arm in a position midway between pronation and supination. Fix the shoulder-cap splint, which should extend well down to the elbow, and then secure both in place with a spiral reversed bandage terminating as a spica of the

shoulder. Finally, suspend the hand and fore-arm in a triangular sling from the neck.

Elbow-joint.—I regard an anterior angular splint as superior to one that confines the fore-arm in an extended position. By placing the fore-arm in the latter position the humero-ulnar angle can be best kept under control; but when it is thus placed, anterior rotation of the condyloid fragments is inevitable. Then, besides, the probability that there will be some impairment of the joint is increased and, if anchylosis should result, the position of the limb will be most objectionable. Besides these objections to the extended position, there is the fact that the angular position is the most comfortable for the patient.

Fore-arm, Shafts of Both Bones.—The prevention of the encroachments of the fragments and of reparative material on the interosseous space, is a matter of great importance, since the functions of pronation and supination are hindered or may be entirely lost. This may be prevented by using an anterior splint with an elevated ridge along its central portion. Some, however, think the influence exercised by pressure upon the deep muscles of both surfaces of the fore-arm by the means of ordinary splints as being equivalent to, if not better for the purposes of separation than specially fashioned splints. At any rate, the splints employed should be a little wider than the fore-arm, and the fore-arm should be placed at a right angle to the arm, midway between pronation and supination. In this manner lateral pressure of the encircling bandage on the fore-arm is prevented by the splints being a little wider, and the position midway between pronation and supination places the bones fartherest apart. The plaster-of-Paris dressing for a fracture of both bones of the fore-arm is very liable to press upon the sides of the fore-arm, tending to cause the fragments to encroach upon the interosseous space.

Colles'.—In reducing this fracture, I am wedded to Levis' formula, which is: first, make hyperextension, to unlock the fragments and relax the dorsal periosteum; secondly, make longitudinal traction to separate the fragments; and, lastly, make forced flexion to get them into position.

CLINICAL NOTES ON OBSTETRICS.

GEO. C. TRAWICK, M. D.

Remember as a good working rule, not to allow a primipara to remain in the second stage of labor over two hours, and a multipara not more than four hours.

One vaginal examination, made deliberately and carefully, is infinitely better than two shorter and less careful ones, and the patient is subjected to less risk of infection.

The Sims picture offers many advantages during delivery. A better view of the maternal soft parts is obtained, the perineum can be better guarded, cleanliness is more easily maintained, and the mother is less aware of exposure.

Do not be too hasty when extracting a breech. Nature allows a considerable time of exposure of the child before danger arises, and a hastily-extracted child may present a fractured bone, which is embarrassing to the accoucher, to say the least.

The use of the forceps on the after-coming head is probably a useless procedure in nine out of ten cases in which it is done. As much traction can be secured on the foetus with the unaided hands, in these cases, as can be gotten with forceps.

Nursing infants may be unable to nurse their own mothers without vomiting the milk, but will be able to take the milk of a wet nurse. Such cases are due to the carrying over into the mother's milk of minute amounts of toxins generated in the system of the mother.

Never allow the on-coming head to be delivered during a pain, if possible to prevent it. When the widest diameter of the head has appeared at the vaginal outlet it should be held there until contraction of the uterus has ceased, and then delivered by flexion. This can be facilitated by pressure on the head, the force being applied externally from a point just behind the rectum.

HISTORY AND BIOGRAPHY

DR. RICHARD DOUGLAS.

The death of our distinguished fellow, Dr. Richard Douglas, cast a gloom over our beloved Southland, and has filled the hearts of the medical profession with grief, and brought sorrow to the homes in which he practiced, so that they all cry with one voice that they shall not see his like again.

But it was not alone in the homes of his patients that his skill and character were prized; the profession he honored was not slow to recognize his peculiar claim to distinction, and years ago had pressed upon his brow all the chaplets of honor that they had power to bestow, and with their appreciative favor he had long since won for himself a place among the foremost surgeons of our common country.

Richard Douglas, the son of Byrd and Sarah Cragwall Douglas, was born and reared in Nashville, Tennessee, his native city, completing his literary education in Nashville schools and colleges, he entered upon the study of medicine in the Medical Department of the University of Nashville from which he received the degree of M.D. in 1881, later completing his course in Jefferson Medical College in 1882. After graduation he returned to Nashville, Tennessee, and entered actively into the practice of the profession to which he devoted the remainder of his very useful life. Not content with his knowledge, but with that avidity which he at all times displayed for more learning, he left soon afterwards and crossed the ocean in search of more, visiting all of the continental hospitals of note, he decided to remain in London and become the private pupil of Mr. Granville Bantock. Returning to Nashville he again entered actively into his practice, which afforded him insufficient outlet for his burning and seemingly inexhaustible energy, and with a desire as keen as his splendid abilities to impart his knowledge, he soon organized a private class for instruction in Gynecology and his course soon became so popular that his call to this chair in the Medical Department of the University of Nashville and Vanderbilt University became imperative. From his former pupil and closest personal friend, Dr. W. H. Witt, whose address upon Dr. Douglas as a teacher, which was delivered at the Memorial meeting of the Nashville Academy of Medicine and Davidson County Medical Society, I quote: "I sat within his voice when he was young and full of health and strength, when he was transported with love for what was at that time a new branch of surgery—whose wonderful development, within his busy life was his constant delight and to which he contributed no little—first and last I have known some good teachers, but when after passing through school and college I saw Dr. Douglas and heard him talk, I was charmed at once at his pleasing personality and his excellence as an instructor. His immaculate dress, his contagious enthusiasm, his spirit of fellowship with the students, aroused at once an interest that was deepened into admiration as we discovered that his worth was not based on such superficial virtues, but that with them he combined in an unusual degree the elements of a teacher. There was first a knowledge of his subject, and, what is characteristic of a great teacher, a knowledge with which he was never satisfied. What he knew positively he taught dogmatically, and with great clearness; but where knowledge was shadowy and theoretical considerations covered medical ignorance, it was

his custom to take students fully into his confidence and acquaint them with the limitations of medical science. Besides a knowledge of his subject, Dr. Douglas had the art of imparting what he knew, a clear voice, an orderly presentment of his subject, a happy mode of illustration and a characteristic habit of repetition all conspired to make him an unusual teacher. In the clinic he also excelled. No one could give the history of an illness with clearer emphasis on the essential points, leading skillfully up to the probable diagnosis, and yet bringing to the students' minds all the possibilities of the case. His intimate acquaintance with medical literature and his personal knowledge of eminent surgeons all over the world were drawn upon for the benefit of his class in the lecture room and in the clinic, and served to assure the student that in every feature his instruction was fully abreast of the times. It would be impossible to estimate the influence of such clear teaching upon the professional lives of the hundreds of young physicians who have come directly under his influence.

"His insistence was always upon a thorough study of the case leading to the diagnosis if possible, and then bold treatment. No shirking, no argument against the plain meaning of the facts, no subterfuges to cover faults in the management of the case. Alertness in diagnosis, promptness in attacking disease were his clinical watchwords. Again, the human side of the man was never kept from his class—to them he was not Professor Douglas difficult to approach, and resenting an expression of friendship. They were made acquainted with his successes and his failures. They saw him as we have seen him—in an ecstacy, or in a gloom, and this very laying bare of his feelings of disappointment or elation made them feel very close to him. He was kind to those in special trouble and in his rather long career as a teacher more than one has had cause to be grateful for acts of kindness and sympathy."

Though a teacher of note, his contributions to medicine were fewer than should have been expected, for a careful search of the literature at hand fails to reveal anything from his pen during the period dating from his return from Europe until 1887, at which time he gives us a splendid article in the New York Medical Journal on "Laparotomy, Practically Considered." During the same year he published a case of "Hemato-salpinx; Laparotomy and Recovery," in the American Journal of Obstetrics and Diseases of Women and Children. In reviewing his paper first mentioned, and following his writings throughout we can not but be impressed by the extreme modesty and love of truth which was his to a remarkable degree. He proceeds in this paper to report his first five cases of abdominal section and halts to put on record one of his especial characteristics, his absolute fidelity to truth. He says: "Were we to place on record only our successful cases our motives might be impugned. In the four cases, as reported above, good fortune has attended all my operations. Not so in this last case, which terminated fatally."

In 1889 appear two papers in the New York Medical Journal; one in July on Hysterectomy, with report of a case, with remarks; the other in the same Journal for December, on "The Complications Occurring in the Clinical History of Ovarian Cysts." The last was read before the Southern Surgical and Gynecological Association, November 13, 1889.

In 1890, The New York Medical Journal, for which he seemed to have preference, published in its July number another article from his pen on Hysterectomy for Edematous Fibroid, which was merely the report of a case with recovery.

During the next four years, from 1890 to 1894, I am unable to find much from his pen, but I am quite sure that he was not inactive, and that the fault is mine, for I have not had the time to make the thorough search which

the subject demands. In this year, however, we are again rewarded with a most admirable monograph on the "Recognition and Management of Wounds of the Urinary Bladder." In this, as in the preceding articles, it admits of no critical analysis in view of the fact that it is so thorough and comprehensive. It was published in the Southern Practitioner in January, 1894. It was during this year that he delivered his presidential address before the Tri-State Medical Society, selecting as his subject, "Responsibilities of the Abdominal Surgeon," to which I have already referred. This address was printed in the Journal of the American Medical Association, May 12, 1894. During October of the same year we find his classical article on "Extra Uterine Pregnancy," read before the Tennessee State Medical Society, and published in the International Medical Magazine of that date.

In November the same year he addressed the Southern Surgical and Gynecological Association and selected for his subject "Acute Peritonitis," which appeared in the American Gynecological and Obstetrical Journal for January, 1895. These two articles from his pen were masterly indeed, and from their wide circulation gave him a position of prominence, and he became recognized as an authority. Resting for a short period he is again awakened by an intense interest in overriding the teachings of physiologists as to the function of the spleen, and in 1896 he reports again to the Southern Surgical and Gynecological Association a successful removal of that organ, and presents a paper unusually complete with historical data upon "Splenectomy Statistically Considered." This paper was published in the Journal of the American Medical Association, April 25, 1896. In 1897 a new and rather neglected field attracted him, and before the American Association of Obstetricians and Gynecologists at Niagara Falls he presented perhaps the most complete and up-to-date monograph on "Cysts of the Urachus," that had ever been written. Resting upon the approbation and applause which he received from this paper, he waited until 1898, when he again appeared before the Southern Surgical and Gynecological Association and presented a rather exhaustive monograph upon "A Study of Retro-Peritoneal Neoplasms, with Special Reference to Diagnosis." This was published in the Journal of the American Medical Association, March 26, 1898.

In February of the same year he delivered a clinical lecture on "Tuberculous Peritonitis," which was published in the Southern Practitioner, and he was then laying the foundation for his more excellent address upon the same subject, which will appear later. That he was deeply interested in the subject of Peritonitis at this time is evidenced by the fact that he selected "Acute General Peritonitis" as the subject for his presidential address before the Southern Surgical and Gynecological Association at Memphis, Tennessee, December 6, 1898.

In 1899 he gives us a report of five cases of "Gunshot Wound of the Abdomen," which report was published March 18, in the Journal of the American Medical Association. Time nor space will not permit me to discuss these cases, but the same truth holds still that he reports them with fidelity to the most trifling detail, and we will see that he is gradually laying a foundation in these operations at the City Hospital for his classical article, which I have already referred to.

After this date, March 18, 1899, we can find no further contribution to medical literature, for he is now doing a greater and grander work, and while it seems that he has dropped from notice, he suddenly offers through his publishers, Messrs. P. Blakiston's Sons & Co., his octavo volume upon "Surgical Diseases of the Abdomen;" which not only served to place him far above the position of an ordinary writer, but spread his fame over Tennessee, the Southland, and America. His publishers inform me by letter received March 8, 1908, that the first edition of this work was soon exhausted, and sold most satisfactorily

in England and Australia. They were also kind enough to forward me hundreds of press clippings, extracted from Journals over our entire land from Maine to California, as well as a number from England, and all of them universally applaud this monumental work.

The careful reader will be struck with the wide range of topics discussed, and even though as the title of his work indicates, it is confined to the diseases of the abdomen, on almost every page will we find him striking out in his comprehensive manner into other departments of medicine, always with a metaphysical acuteness equalled only by his immense grasp of the fundamentals. Alike remarkable for his profound analysis, constructive ability and power of lucid and forcible statement, Dr. Douglas was well equipped for the work with which he honored medicine. When we remember the state of his health, the exactions of a large and trying practice, we wonder that he was bold enough to undertake so difficult a task. It required untiring work and indefatigable nature, but such were his endowments.

Previous to the publication of his book, his contributions to medical literature were further apart than would have been expected of a man with his natural ability in this particular field, coupled with unquestioned opportunities. I believe I would do his memory an injustice should I not attempt to explain or give a reason for the scarcity of his medical writings. I refer particularly to journal articles.

It was because he was a man possessing to an unusual degree an excess of modesty, and it was his custom to be his own severe critic; an article to which his name was to be attached must possess a standard of merit (measured by his own critical judgment), or else it would be shelved for further revision or destroyed, and many, many hours of labor were so wasted. His excess of modesty caused him to shrink from publicity, and time and time again he would refuse to have a paper published because he did not think it was as good as he was capable of making it. His standard was to "have a message and deliver it," and unless the subject presented manifold complications, he derived no pleasure in trying to solve it. He had no time for the commonplace in medicine, always bending his energies against the real problems of medical and surgical science.

His modesty not only reduced the number of his writings, but often would he give credit for some original observation to another.

In one instance, with which I happen to be familiar, he did so, in his magnificent paper on "The Etiology, Prognosis and Indications for the Surgical Treatment of Tuberculous Peritonitis," which he read May 17, 1906, before the National Association for the study and prevention of Tuberculosis at Washington, D. C., printed in the American Journal of the Medical Sciences. Here he deliberately attributes to another an observation, which he told me he himself had made. What a pity that we cannot have more men with the modesty which this example typifies! How easy would it have been for him to say, "Under my direction a series of experiments were undertaken to determine," etc., but not a word of self or me or I; modestly he assumes the position of a mere statistician and throws away whatever glory may have come from such an observation.

It is quite fitting that his last epoch-making article should have been read before the Nashville Academy of Medicine. It was a special address delivered January 1, 1907, and his subject was "The Treatment of Acute Diffuse Peritonitis." The address was published in the Medical Record of February 23, 1907, and was the cause of many highly complimentary letters to Dr. Douglas from eminent men throughout the country.

As a man he was of simple habits, too enthused with professional labors to indulge in social exercises. He formed few friendships, but those with fervor and loyalty. He attend-

ed medical organizations because he loved to learn and to instruct, but above all else he despised medical politics. It is needless to say that his election to the presidency of nearly all the Associations to which he belonged was always over his protest.

His irritability, his temper, and often his bitter hatred over real or imaginary wrongs were his greatest faults; but the profession and the people loved him, not for his defects, but in spite of them.

Dr. Douglas was a member of the Nashville Academy of Medicine, British Gynecological Society, of the American Medical Association, ex-president of the Southern Surgical and Gynecological Association, ex-vice-president of the American Association of Obstetricians and Gynecologists, and ex-president of the Tri-State Medical Society of Alabama, Georgia and Tennessee. He was also one of the five American delegates to the International Medical Congress in Madrid, in 1903.

Rarely gifted with pen and tongue, forceful, original, courageous and cheerful, kindly and genial, loving his fellowman and beloved, esteemed and respected by a multitude of admiring friends, he was taken from us in the meridian of life, when he showed every promise of adding further laurels to his crown of glory and shedding still more honor upon this our Southland.

PERRY BROMBERG, M.D.

Obituaries

The staff of the SOUTHERN MEDICAL JOURNAL, with deep regret, notes the death of that eminent surgeon, Dr. A. M. Cartledge of Louisville, Ky., Professor of Gynecology and Abdominal Surgery in the Louisville Medical College; Ex-President of the Medico-Chirurgical Society; Ex-President of the Southern Gynecological Association. He was one of our honored collaborators, and from his ability as a writer and thinker this staff expected many valuable contributions to this Journal. The death of Dr. Cartledge, which took place on May 11, 1908, is not only a loss to Louisville and to this Journal, but is a distinct loss to the profession of the United States. His death is especially sad in the fact that he was just in the prime of his usefulness, and was one of the shining lights which had reflected so much credit upon Southern medicine. This Journal sends its sincerest sympathy to his family and friends in Louisville.

It is with extreme regret that we have to report the death of Dr. T. E. Schumpert, of Shreveport, La., who was a collaborator of this Journal, and whose standing as a gentleman and professional man was second to none in the South. In his death the old adage was proven, in that death loves a shining mark. The Journal extends its sincerest sympathy to his family, in this, their great loss, realizing that the Journal has also lost not only a staunch friend, but a man who would have contributed much to its success.

ABSTRACTS OF CURRENT LITERATURE

MEDICINE

THE USE AND ABUSE OF DIGITALIS.

—*The American Journal of Medical Sciences.* (Vol. 135, June, 1908.)

Janeway. Digitalis, our mainstay in treatment of cardiac diseases. The causes for failure in the employment of digitalis, in the order of their frequency and importance are:

1. The use of inefficient preparations.
2. Use in unsuitable cases.
3. Improper dosage.
4. Improper methods of administration.
5. Neglect of other necessary therapeutic measures.

In order that we may be sure of giving efficient digitalis the following conditions must be followed:

1. The leaves must be from plants of the second year's growth picked at the beginning of efflorescence, freed from stalks and carefully dried.

2. The dried leaves must be kept absolutely dark and free from moisture in sealed tin or glass containers, and for not more than one year.

3. The preparation dispensed must be freshly made from these leaves in exact accordance with the method prescribed by the dispensatory.

Specify preparation to be made from Allen's English leaves. The worst digitalis preparation is an infusion made by diluting the fluid extract. Other inefficient forms are the tablets and pills containing digitalis. Under proper conditions the infusion, tincture, and powdered leaves may be considered trustworthy. One of the most deplorable conditions is the combinations of heart tonics, usually with nitro-glycerine, in tablet form. For hypodermic use a good tincture is, perhaps, most certain in its action.

The large muscular rings surrounding the mitral and tri-cuspid orifices are especially endowed with tone. The digitalis series clearly exert a direct chemical effect on the heart muscle itself, and indirect effects by vagus stimulation and vasoconstriction. The direct action of digitalis is to increase contractility, tone and irritability. Through direct stimulation of vagus centre, digitalis acts to slow the heart. Such inhibitory action depresses tone as well as rate, and for this reason it is not a good remedy for tachycardia when no ventricular insufficiency exists.

Seldom need to use more than two drams of a good infusion and larger doses of a poor preparation are mainly successful in deranging digestion. Digitalis should never be pushed for more than a few days without careful records of heart as well as pulse rate, and of amount of fluids taken and urine passed each 24 hours, with these precautions the dosage of an infusion may be increased from two drams to half an ounce, and of the tincture from 10 to 20 minims every four hours. Any marked decrease in urine is a danger signal not to be neglected, for, with diminished excretion of the drug, cumulative effects may supervene. Marked slowness and irregularity of heart, evidence the second stage of digitalis poison. Increased rate, with irregularity, marks the onset of third stage of poisoning.

One common error is persistence in administering it by the mouth when frequent vomiting shows there is little hope of absorption from the stomach. Two other paths of absorption are rectum and the subcutaneous tissue.

More than digitalis is needed in most heart cases. First, in the list is rest. Another essential in dropsical patients is a dry diet. When anemia is marked give iron, for anemia itself causes loss of tone of the heart muscle. Finally, one of the newly-heralded therapeutic methods is psychotherapathy, cheerfulness, courage, hope. (O. N. B.)

OBSTETRICS AND GYNECOLOGY

A CASE OF INTRA-UTERINE DEATH OF THE FOETUS OCCURRING IN SIX CONSECUTIVE PREGNANCIES: WITH OBSERVATIONS UPON THE IMPORTANCE OF THE EXAMINATION OF THE FOETAL TISSUES FOR SPIROCHAETA PALLIDA.

Herbert Williamson and Eardley L. Holland, in the *Journal of Obstetrics and Gynecology of the British Empire,* report an interesting case in which an investigation was made as to the presence of spirochaeta pallida in a stillborn child. This case is reported as a contribution towards the pathology of intra-uterine death. Syphilis is justly regarded as one of the commonest causes, and the case in point demonstrated that even in the absence of the history of infection or of any clinical manifestations of the disease in the parents, syphilis may be responsible for the death of the child. The child was premature, presumably of about 36 weeks gestation and macerated. Unfortunately the placenta had been destroyed and was not available for examination. The pathologist reported that spirochaeta were distributed throughout the whole section of the liver, more or less uniformly, but in certain small areas were grouped very closely together. Whether these areas correspond to the site of the blood-vessels it was difficult to say, for the method employed did not admit clear differentiation of the tissues. It must be remembered that all the blood of the foetal circulation passes through the liver and the presence of spirochaetes in or around the hepatic capillaries is not surprising. The spleen showed spirochaetæ in the same abundance as those of the liver, but were distributed more uniformly, and did not clump as in the latter organ.

No spirochaetes could be detected in the sections from the umbilical cord. The evidence obtained from the study of this dead foetus appears to prove conclusively the presence of a syphilitic infection even in the absence of any clinical manifestations of syphilis and in spite of a negative history.

In addition to the case just recorded the authors examined at Queen Charlotte's Hospital the organs of seven macerated foetuses and found the spirochæta in great abundance in six. Of these, in five there was a history of syphilis in the parents; in the single negative case there was no such history. The organs examined were the liver, spleen, kidneys and lungs, and the spirochæta was present in each case in all of these organs, but most abundantly of all in the liver and the lungs. In the placenta they never could find it. Bab is quoted as having examined a large amount of material in very great detail, and found the spirochæta in the various organs in the following proportions: Lungs, 87 per cent.; pancreas, 80 per cent.; skin, 66 per cent.; suprarenal, 64 per cent.; spleen, 62 per cent.; liver, 59 per cent.; kidneys, 54 per cent. He found it in the umbilical cord in 9 per cent., but never in the placenta. The constant presence of the spirochæta pallida in the organs

of these congenitally syphilitic foetuses, and the absence of all other organisms, is a remarkably strong evidence for the specificity of the spirochaeta pallida as the infective agent in syphilis. The authors believe, therefore, in the routine examination of the tissues of premature stillborn children and consider it a valuable method of elucidating the cause of intra-uterine death in certain obscure cases, and that we may be afforded a clue to the line of treatment by which we may prevent a recurrence of such a disaster.

A STUDY OF THE CONDITIONS THAT REQUIRE THE REMOVAL OF THE CHILD FROM THE BREAST.

The following conclusions are drawn from an extensive paper on this subject by Charles B. Reed (*Surgery, Gynecology and Obstetrics,* May, 1908):

The first part of the work deals with the effects of drugs upon lactation.

This investigation proves a source of considerable surprise and of great interest. The scarcity of clinical reports in literature has been no less surprising than the almost valueless character of those found. It seems probable that alcohol, opium, chloroform, ether, thyroid extract and lead should be used with extreme care in a nursing woman until their status is determined; that zinc apparently has no effect, while all the others investigated, namely, atropine, arsenic, antipyrine, acetic acid, bismuth, brom. potassium, balsam of copaiba, castor oil, copper, chloral, iodine and its compounds, mercury, phenacetine, quinine, salicylic acid, and senna, pass over in small quantities, but very rarely to a degree injurious to a sensitive babe. Hence it is practically never necessary to remove the child from the breast on account of drugs administered to the mother. That this position is correct is substantiated by the fact that cases occur so rarely in the literature. A logical inference from the above data may also be drawn that, owing to the total absence of transmission, or owing to the impossibility of satisfactorily measuring the amount transmitted, it is never desirable to attempt to medicate the child by means of drugs administered to the mother.

The second portion of the article deals with a number of anatomic, physiological and pathological conditions that prevent the nursing of the babe.

From his study of all the materials at hand, Reed concludes that the woman who is abandoned to the care and nursing of her babe without expert advice, quite commonly gets bad results and not infrequently fatalities. The increase in the weight of the babe should be regularly followed on the scales and accurately recorded; the stools examined frequently; and when cutaneous manifestations appear, such as erythema, even though slight, the milk of the mother should be analyzed and her physical condition investigated.

We find that while the return of the menses need not necessarily contraindicate nursing the babe, yet if the child begins to show digestive disturbance and intestinal irritation, this source of trouble, so often overlooked, should be taken into consideration, and its effect carefully estimated.

The same deduction must be made in regard to mental disturbance during lactation, while albuminuria and cardiac lesions, unless quite unimportant, will more often require removal of the child on account of the mother rather than the child. Removal from the breast is imperatively demanded for the sake of mother or child, or both, when lactation is complicated with mania, or epilepsy, except in very light and infrequent attacks.

Salge maintains that a mother with scarla-

tina, measles, and diphtheria should continue to nurse her babe and claims that the ingestion of such a nourishment will render the babe forever immune to these diseases.

Considering the ease with which streptococci enter into and disseminate themselves through the blood, this theory has very serious objections, and would appear to be highly dangerous. The risk in nursing a child under such conditions is far more hazardous than any possible benefit that could be secured. Further, it is not probable that immunity to diphtheria would be conferred for a longer period in any case than when secured by injection of antitoxin—say from six to eight weeks.

Nephritis, serious and obstinate erosion and fissure of nipple, Paget's disease, scirrhus, sarcoma, or abscess of the breast, together with local or systematic tuberculosis, syphilis, erysipelas, acute articular rheumatism, acute pulmonary or pleuritic affections, and all infectious and contagious diseases, osteomalacia, and all puerperal infections that are not mild and transitory in character, as a rule, demand the absolute separation of mother and child.

MILLER.

A CASE OF PUERPERAL ECLAMPSIA TREATED BY RENAL DECAPSULATION.

By R. J. Johnstone, *The Practitioner,* June, 1908.

A primipara, age 24, passed through a normal gestation to term. A short time after onset of labor, she had an eclamptic seizure. As the os was sufficiently dilated, forceps were at once applied, and delivery completed within thirty minutes after the convulsion. Two more fits occurred during the third stage, and shortly after the delivery of the placenta, the fits became established, with a periodic recurrence of twenty minutes. Morphia was administered, purgation and diaphoresis instituted. Six hours later, patient was comatose and cyanotic. Morphia was repeated, gr. 1-3 hypodermically, and five pints of saline were given subcutaneously. Chloroform was administered a short time before each convulsion was due, but as it did not serve to prevent their occurrence, it was discontinued. The right kidney was decapsulated eight hours after onset of eclampsia, the operation lasting twenty minutes, and the patient had a convulsion while on the table. In the two hours following she had three more, and during the twenty-four hours she had altoegther, forty. Twenty pints of saline were given in all in twenty-four hours.

The number of fits, and the duration of unconsciousness mark the case as a severe one.

Mr. Johnstone attributes to the decapsulation of the kidney the credit for the recovery in this case. After discussing other remedial agents used, he reports the fact that before delivering this patient with forceps, a small amount of urine was removed from the bladder with a catheter, and it was found to be full of albumen. During the eight hours between the onset of paroxysms and decapsulation, only one and one-half ounces were secreted. In the ensuing two hours, twenty ounces were secreted, in the next fourteen hours, twenty ounces, and in the eight and one-half hours following, thirty-two ounces. He thinks there is no doubt but that the operation succeeded in re-establishing the secretion of the kidneys. The injection of saline aided very much in the flushing out.

The question as to the after-result of decapsulation on a healthy kidney he thinks need cause no apprehension, as untoward results have never followed the fairly wide stripping of the capsule which is a feature in nephropexy. While the value of decapsulation is not proven, he recommends its consideration in any severe case of sclampsia which is not yielding readily to other remedies. (G. C. T.)

GENITO—URINARY AND DERMATOLOGY

TREATMENT OF ULCERS.

Bertrain C. Stevens, *Practitioner* (Lon.), Aug., '07, page 290.

Treatment of tuberculous ulcers and ulcers due to local causes is discussed, syphilitic and others being omitted. The main object in presenting the article is to advocate the use of calcium iodide internally and the application of Robinson's (*Lancet*, 1897) decalcified cancellous bone externally to the surface of the ulcer.

First, reduce all ulcers to simple healing ulcers by curetting; use of caustics, as copper sulphate, silver nitrate and wet dressings of zinc chloride or sulphate solution with or without oiled silk as the ulcer may be dry or moist.

Second, skin the ulcer over. He enumerates the ordinary methods and then concludes with the bone dressing for the clean, healthy ulcer, as follows:

It acts by mechanical advantages, no inherent virtue in itself. Being light and porous it fills with lymph secreted by the ulcer or wound, and these lymph cells act as little beds of nourishment for the granulations, and draw on the young capillaries and so promote healing.

To use the bone, swab the ulcer with hydrogen peroxide, then slice the bone and lay the pieces over the ulcer. Over the dressing then place a piece of boracic lint wrung out of hot water, and apply wool and a crepe bandage. If the ulcer is very large, only place the pieces of bone around the edge and dress as before. If the ulcer appears very dry, use oiled silk; if moist, omit it. The bone dressing must be changed every three or five days, according to the discharge. At first the bone will be nearly all dissolved, but later on it remains clinging to the edges with the blue line of epithelium underneath.

The author considers the bone dressing a scientific, useful and fascinating application to ulcers and open wounds generally.

He regards calcium iodide as a rule the best internal treatment in conjunction with tonics, etc. (J. M. K.)

TREATMENT OF CHILBLAINS.

F. Gardner, *Practitioner* (Lon.), Feb., '08, page 251.

Chilblains are neglected — Quackery is a plant which grows on neglected soil.

Patient's general condition and circulation should be carefully examined and proper treatment administered.

Local Treatment.—For early stage, warm, loose clothing. Two drugs of pre-eminent value—Ichthyol and Formalin. Ichthyol reduces congestion. Ten to twenty per cent. in Lanolin is bound to the parts at night, and worn in the form of a plaster during the day. Adrenalin and chloretone are not so valuable.

Formalin is an astringent, antiseptic and more effective and lasting in its results; and must be used with great care, or the skin will become very hard and will crack. The application of 10 to 50 per cent. in Lanolin may be used as a rule, but the pure solution may be used on coarse skins.

Begin with Ichthyol and finish with Formalin.

He also recommends the Faradic bath, *x*-rays and high frequency.

If treatment fails, think of Lupus Erythematosus. (J. M. K.)

SURGERY

SURGICAL ASPECTS OF VARIX OF THE LOWER LIMBS.

By Raymond Russ, M.D., in *Surgery, Gynecology and Obstetrics,* July, 1908.

Varix is to be distinguished from simple dilatation. The former only is discussed. There are from one per cent. to four per cent. of applicants for police and army service, showing the condition. The cause of the condition is not fully settled. Most cases of the author's one hundred are males. This is substantiated by many other writers. Constipation and abdominal cysts and tumors have influence on etiology. Seven of the patients first noticed the veins after fractures in the lower extremities. Russ thinks heredity is overestimated. In only three instances has he treated more than a single member of a family. Pregnancy produces dilation, which usually disappears. If there are numerous conceptions varix often results.

Trendelenburg's test is advised as the most valuable physical sign from the surgeon's standpoint. Often it is not visibly present, but on release of pressure at saphenous opening, a thrill will be felt, showing valvular insufficiency.

Standing for a long time or habitually sitting long in one position, especially if the muscles of the arms are brought into play, bicycle riding, all are frequent causative factors.

The age of onset in the majority is between the 20th and 30th years. Average of Russ' cases was 36. Venous supply is much more than the average demand. The contracting muscles assist flow of blood in deep veins, cessation throws extra work and pressure on superficial ones, as seen in the athlete after running. The long saphenous valves are always intact in the fetus and gradually become insufficient. At twenty-five, seventeen per cent., at fifty, forty per cent., at seventy, eighty per cent show insufficiency. Internal saphenous averages three to nine valves; it has superficial branches and deep anastomosis, and it communicates freely with the external saphenous.

Clinical grouping: (1) Limbs enlarged, possible ulcers, edema, eczema and pigmented spots, the long vein shows little or no varicosity; there are small dilated veins on dorsum of foot and on ankle and a few clumps between ankle and knee at the point of anastomosis, with deep veins. (2) Marked varix, little or no enlargement. Internal saphena prominent, especially in the leg, with hard, tortuous branches, phlebitic spots, and phleboliths present—Trendelenburg's test present. (3) Isolated patches of varix in thigh and leg, little or no enlargement. Internal vein may be palpable at points. Trendelenburg's test absent. If the deep veins are involved, as they frequently are, in 1 and 3, the general and skin appearances may be large relative to the dilation of the superficial veins. The most obstinate ulcers often occur with little surface dilation.

Symptoms are pain increasing during the day, numbness, cramps, itching, edema, and tropic changes, skin little resistant to infections and may be thick and brawny, muscles relaxed and atrophic, flat-foot is often seen. Violent exercise makes them worse; moderate exercise is helpful unless complications are present.

Treatment in group 1, operation is valueless, here only palliative measures are helpful. In group 2, is the best field for operation, which is best done by total extirpation of the long saphenous; seventy-five to eighty per cent. are cured.

In group 3, operation is precluded except where iminent rupture is feared, or where the pressure of the varicosities are annoying the patient. (W. A. B.)

ON THE PATHOGENESIS AND OPERATIVE TREATMENT OF PROLAPSUS.

J. Rotter, *Deutsche Medizinische Wochenschrift,* April 16, 1908.

Rotter gives the factors that favor the development of prolapsus as follows: The pelvic floor is relaxed, and the sphincters become so loose that the two fingers can often easily be introduced. The posterior limit of the anus is displaced backwards straightening out the lower end of the gut, bringing the outlet into the line of the greatest downward pressure and at the same time making it impossible for the anterior wall of the gut to be forced against the posterior in such a way as to throw the center of pressure behind the outlet. When the anus is thus placed in the line of pressure the gut easily and gradually develops a prolapse. Relaxation of both fascia and muscles favor the condition and a congenitally low Douglas' cul-de-sac has been proved to be an important etiological factor.

Removal of the deeply situated cul-de-sac, rectopexy, colopexy the author condemns as being failures in too many cases or impracticable. Thiersch's method of narrowing the sphincter by using a wire ring has given 27 cures out of 31 cases. It is inconvenient for the patient, as the ring must be carried from one year to 18 months.

Verneuil's method and the modification by Helferich, incises the mucosa or skin, and narrows the sphincter at the posterior limit. Hoffman exposes the posterior border of the sphincter and the levator ani: with a hook he pulls the sphincter forward and places sutures from levator and through the sphincter and out again through the levator ani on opposite side, when the sutures are drawn the posterior margin is held in its new and normal position.

Rehn & Delorme's method, Rotter thinks, the best of any. Traction sutures are placed at the summit of the prolapse. Then the surface is cut circular at the mucocutaneous juncture and the lining of the gut dissected up as in Whitehead's operation for hemorrhoids: at the upper limit this is cut off. Five or six longitudinal sutures are stitched into the raw surface and tied, thus creating a buttress and a narrowing of the outlet at once. Suture the mucous membrane to the skin. The bowels should not be moved until the tenth day, when union will have occurred. Out of 24 cases so treated, there was only one partial failure. (W. A. B.)

A SYMPTOM OF PERFORATION OF THE APPENDIX.

M. Pierre Delbet in *La Presse Medicale,* June 3, 1908, gives an interesting lecture on "A Symptom of Perforation of the Appendix." He advises against operation during the attack unless there is a special reason. He describes the case: "In fact, the general condition of this boy appears excellent, his countenance is good, without emaciation, with no sinking of the eyeballs, no pinching of the nose, and even without pallor. The voice is not broken; he talks easily and relates with a strong voice his entire history. His pulse is good and 100 per minute. The abdomen is neither distended nor retracted. There is nothing to cause suspicion of generalized peritonitis, and yet I am convinced that the appendix is perforated, so convinced that my lecture will last only long enough to prepare for operation. That will be long enough to give the reasons for my diagnosis."

The appendix is not attached to the cecum at McBurney's point and the fact that the pain is not at that point should not mislead the diagnostician. The appendix is usually lower, at the level of the superior iliac spines.

He calls this the appendicular point, and here is often more sensitiveness than at McBurney's point. It is so in the case in question, although the usual point of tenderness

is at McBurney's point. Delbet thinks the reason for this usual tenderness at McBurney's point is secondary infection of the inter-ileo-cecal glands.

It is rare that a first attack of appendicitis perforates. This is not the first attack in this case. The present attack began suddenly night before last at 11:30 o'clock, and he entered the hospital yesterday noon, when the best treatment was instituted, viz: rest, diet, ice, with the usual effect—relief. Last night without imprudence, with no cessation from the above named treatment, the patient was seized with a new violent attack. It is now relieved. This is the symptom—return of the pain in the form of a crisis, in spite of treatment—which makes me give the diagnosis of perforation.

Delbet then cites several similar cases, and discusses the leucocyte count, and the proper time to operate in case there is no perforation, concluding that it is better to wait usually till the interval. The factor that determines when the attack has subsided is the return of the temperature to normal. Operation revealed a perforated appendix.

(W. A. B.)

EYE, EAR, NOSE AND THROAT

HEMORRHAGE FOLLOWING QUINSY.

Ligation of Common Carotid Artery; Recovery. With a Study of Fifty-one Cases of Hemorrhage in Connection with Pharyngeal Suppurations. By James E. Newcomb, M.D., New York City, in London *Journal of Laryngology, Rhinology, and Otology.* June, 1908.

Dr. Newcomb reports case of stout, plethoric German, aged 55. Good liver, and free consumer of alcohol. Vessels showed distinct arterio sclerosis. No renal trouble. Had numerous quinsies extending over period of 40 years. No previous attack for ten years. On Feb. 3, 1908, had an attack affecting both tonsils. On Feb. 6 pointing was observed far out in soft palate on left side, which was incised by Dr. McBarron. At a depth of 3-8 of an inch a cavity was entered, and a drachm of pus voided. Thirty-six hours later bleeding recurred from incision distinctly arterial in color. Dr. Newcomb was called in with Dr. McBarron, and the cavity packed with gauze dipped in aceto-tartrate of alum. Bleeding did not recur for thirty-six hours, but was more persistent and increased in amount. Packing and ice internally and externally did not control the hemorrhage and resort was had to ligation of the left common carotid. Bleeding ceased instantly and never returned. Patient made good recovery and there was no disturbance of cerebration.

He reports fifty additional cases from the literature. There were eleven cases of spontaneous opening of the abscess, followed by immediate hemorrhage with seven deaths and four recoveries. In all four recoveries ligation of common carotid was resorted to. In the seven fatal cases no operation was done. The cause of the fatal hemorrhage in each case was ulceration of internal carotid.

There were fifteen cases of spontaneous openings of the abscess with secondary hemorrhage, followed by eight deaths and seven recoveries. In the eight fatal cases there was no operation. In the seven recoveries there were four ligations.

There were seven cases of abscess opened by incision with immediate hemorrhage, which were followed by four deaths and three

recoveries. In the fatal cases two had the common carotid ligated. In one the fatal hemorrhage occurred on the 28th day in the other the ligature did not control the hemorrhage. In the three recovered cases ligation was performed in two.

There were eight cases of abscess opened by incision followed by secondary hemorrhage, with four fatalities and four recoveries. In the four fatal cases there was ligation of the common carotid in three. In the four recovered cases hemorrhage was controlled by ice, rest and packing without ligation.

In five cases of hemorrhage following retropharyngeal abscess there were two deaths and three recoveries. In the three recoveries there were two ligations and none in the two fatal cases.

There were three cases of hemorrhage in connection with scarlatinal suppuration, with one death and two recoveries. No operation in fatal case; ligation of common carotid in one of the recovered cases.

There were two cases of gangrenous tonsilitis with spontaneous opening and death in both cases. No operation was done.

Of the 51 cases reported there were 23 recoveries and 28 deaths, a mortality rate of 54.9 per cent. The common carotid was ligated 16 times with 11 recoveries and five deaths. The external and internal carotid were ligated once and the external, internal and common carotid were ligated once, with recovery in both cases.

Autopsy records were given in thirteen cases. The bleeding came from the external carotid in two, ascending pharyngeal in one, "carotid" in one, lingual in one, at the bifurcation in one, facial in one. The other cases were from internal carotid. In ten cases in which no autopsy was held the blood was supposed to come from internal carotid.

(M. M. C.)

BOOK REVIEW

A Text-Book of Minor Surgery. By Edward Milton Foote, A.M., M.D., Instructor in Surgery, College of Physicians and Surgeons (Columbia University); Lecturer on Surgery, New York Polyclinic Medical School, etc.

This book, as its name indicates, is not intended to cover the field of surgery, but to give to the working practitioner a condensed and modern understanding of the more common surgical conditions he will be called upon to attend. The work does not confine itself to the treatment of the lesser ailments, but gives a brief synopsis of the clinical manifestations of many diseases heretofore found only in larger and more exhaustive works on surgery. It touches very briefly on all of the practical branches; Gynecology is given about five pages and the subjects discussed are Old Lacerations of the Perineum, Prolapse of the Uterus, Fistula of the Vagina, Adhesions of the Clitoris, Imperforate Hymen and Stenosis of the Cervix. Diseases of the Rectum, Genito-Urinary Organs and Dermatology are each discussed very briefly from the surgeon's viewpoint.

The book offers no pathology and presents no new material. It abounds in photographs taken from the author's extensive practice, which are valuable in fixing ideas of pathological conditions. The chapter on bandaging is especially to be commended. The useless repetition found all through the work we hope will be corrected in future editions.

(P. B.)

BOOKS RECEIVED

Medical Gynecology. By Howard A. Kelly, A.B., M.D., LL.D., F.R.C.S. (Hon. Edinb.). Professor Gynecology and Obstetrics in the Johns Hopkins University, and Gynecologist to the Johns Hopkins Hospital, Baltimore. Pp. 662, 163. Illustrated for the most part by Max Broedel and A. Horn. Cloth, $6.00. D. Appleton & Co., New York and London. 1908.

Text-Book of Minor Surgery. By Edward Milton Foote, A.M., M.D., Lecturer on Surgery New York Polyclinic Medical School. Illustrated by four hundred and seven engravings from original drawings and photographs. 752 pages. Cloth, $5.00. D. Appleton, & Co., New York and London.

Text-Book of Surgical Anatomy. By William Francis Campbell, M.D., Professor of Anatomy at Long Island College Hospital. Octavo of 675 pages, with 319 original illustrations. Cloth, $5.00 net; Half Morocco, $6.50, net. W. B. Saunders Co., Philadelphia and London, 1908.

Bier's Hyperemic Treatment in Surgery, Medicine and All the Specialties. A Manual of Its Practical Application. By Willy Meyer, M.D., Professor of Surgery at the New York Post-Graduate Medical School, and Hospital; and Professor Dr. Victor Schmeiden, Assistant to Professor Bier at Berlin University, Germany. Octavo of 209 pages, illustrated. Cloth, $3.00 net. W. B. Saunders Co., Philadelphia and London, 1908.

The Border-Land of Epilepsy. Faints, Vagal Attacks, Vertigo, Migraine, Sleep Symptoms, and their Treatment. By Sir William R. Gowers, M.D., F.R.C.P., F.R.S.; Hon. Fellow R. Coll. Phys., Ireland. 120 pages. Cloth, $3.00. P. Blakiston's Son & Co., Philadelphia.

State Board Questions and Answers. By R. Max Goepp, M.D., Professor of Clinical Medicine at the Philadelphia Polyclinic. Octavo volume of 684 pages. Cloth, $4.00; Half Morocco, $5.50 net. W. B. Saunders Co., Philadelphia and London, 1908.

Treatment of Disease. A Manual of Practical Medicine. Second Edition. By Reynold Webb Wilcox, M.A., M.D., LL.D., Professor of Medicine at the New York Post-Graduate Medical School and Hospital. Octavo; xxiv plus 932 pages, illustrated. Cloth, $6.00 net; Half Morocco, $7.00 net. P. Blakiston's Son & Co., Philadelphia.

Manual of Fever Nursing. By Reynold Webb Wilcox, M.A., M.D., LL.D., Second Edition. Revised; Illustrated; 229 pages. Cloth, $1.00, net. P. Blakinston's Son & Co., Philadelphia.

Diseases of the Breast: With Special Reference to Cancer. By William L. Rodman, M.D., LL.D., Professor of Surgery in the Medico-Chirurgical College of Philadelphia. With 69 plates, of which 12 are printed in colors, and 42 other illustrations; 385 pages. Cloth, $4.00. P. Blakiston's Son & Co., Philadelphia.

Manual of Orthopedic Surgery. By Augustus Thorndike, M.D., Assistant in Orthopedics at the Harvard Medical School. 400 pages, with illustrations. Full limp leather, gilt edges, round corners; $2.50. P. Blakiston's Son & Co., Philadelphia.

Text-Book of Diseases of the Skin. By Arthur Van Harlingen, Ph.B. (Yale), M.D. Fourth Edition, thoroughly revised and rearranged, with 102 illustrations; 482 pages. Cloth, $3.00. P. Blakiston's Son & Co., Philadelphia.

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